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DEFECTS IN VISUAL FIELD OF ONE EYE ONLY IN PATIENTS WITH A LESION OF ONE OPTIC RADIATION

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Lesions of the optic radiation cause contralateral homonymous defects in the fields of vision. The defect may be quadrantic or hemianopic, with or without sparing of central vision. Sometimes, however, careful perimetric determinations reveal partial and unpaired blind areas, i. e., a defect in one section of the field of vision of one eye but not in the corresponding field of the other eye. The commonest type of unpaired defect in cases of lesions of the optic radiation is known as the temporal crescent or half moon defect. The terms temporal crescent and half moon have reference to either one of the two areas which are temporal and peripheral to the binocular field of vision (fig. 1). Each of these areas is projected on the retina of one eye only (on the nasal retina) and therefore represents the uniocular field of vision. It corresponds to the phylogenetically oldest monocular vision in primates.

ANATOMY OF THE FIBERS OF THE TEMPORAL CRESCENT IN THE VISUAL PATHWAYS

Brouwer and Zeeman [1] demonstrated that after destruction of the peripheral portion of the nasal retina there was degeneration of a bundle of fibers located in the median portion of the optic nerve that crossed in the optic chiasm to the median part of the optic tract and spread downward ventrally into the lateral geniculate body (fig. 1). Wilbrand [2] showed that this small bundle was located in the ventrolateral portion of the optic chiasm and seemed to contain only crossed fibers.

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1. Brouwer, B., and Zeeman, W. P. C.: The Projection of the Retina in the Primary Optic Neuron in Monkeys, Brain **49**:1, 1926.

2. Wilbrand, H.: Schema des Verlaufs der Sehnervenfasern durch das Chiasma, Ztschr. f. Augenh. **59**:135, 1926.

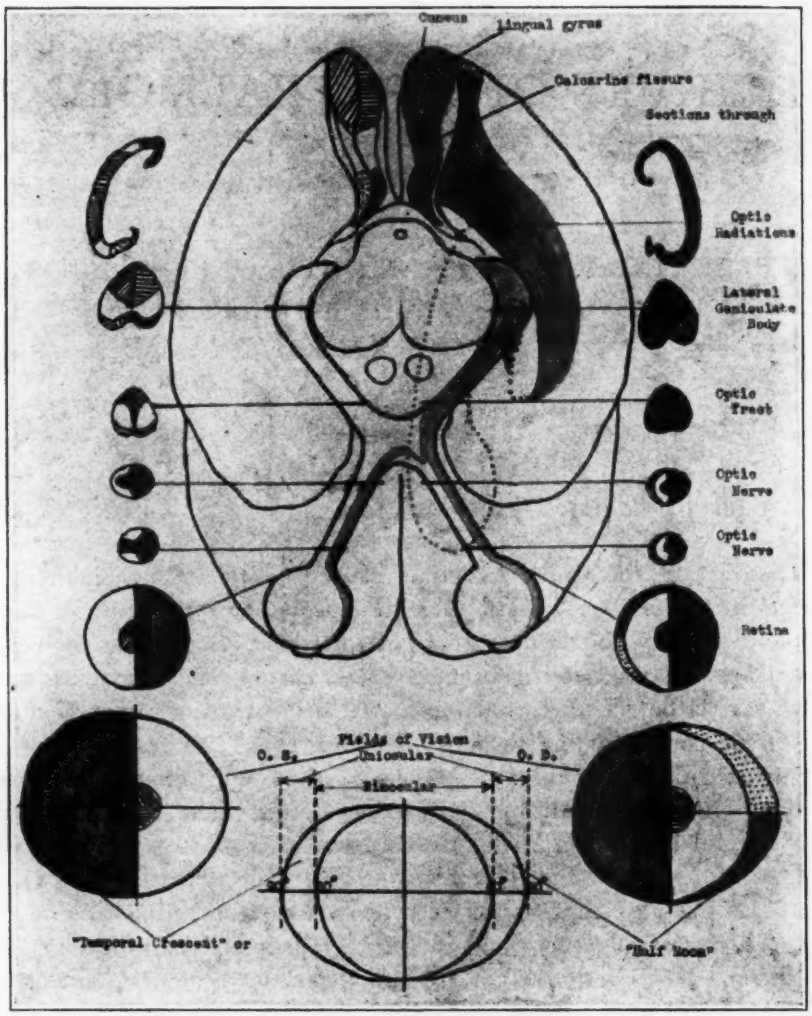

Fig. 1.—Schema illustrating the probable course of the afferent visual pathways at different levels from the retina to the occipital cortex and their projected visual fields. Solid black represents the inferior, and the lighter shade the superior, temporal crescent of the left field of vision; the heavy dots represent the inferior, and the lighter dots the superior, temporal crescent of the right field of vision. These are projected from the corresponding points of the nasal retina of each eye. From the nasal retina the fibers may be traced through the mesial part to the optic nerve, crossing completely in the chiasm to the ventral portion of the opposite optic tract and lateral geniculate body, the mesial portion conducting the fibers for the superior, and the lateral portion the fibers for the inferior, part of the crescent. In the optic radiation the fibers for the superior, and those for the inferior, portion of the crescent separate and course along their respective quadrants at opposite edges of the radiation. The fibers come together again in the most anterior part of the area striata, the fibers for the superior part of the crescent terminating in the cortex above, and those for the inferior part of the crescent in the cortex below, the calcarine fissure. The solid shaded areas represent the binocular left homonymous field of vision. The horizontal lines represent the lower macular, and the diagonal lines the upper macular, field of vision.

The course of this bundle of fibers beyond the lateral geniculate body was ascertained by Poliak [3] in 1932. Pfeiffer [4] in 1925 suggested that these fibers were located in the ventral rim of the optic radiation. Traquair [5] and Wilbrand [2] opined that the fibers for the crescent occupied the most mesial portions of the radiations. While Pfeiffer believed that all the fibers reached the occipital cortex by passing the posterior horn from below, Poliak [3] found that they reached the cortex from above and below (fig. 1). In the cortex of the occipital lobe, the most anterior portion of the area striata harbors the projected crescent of the "cortical retina," the upper half of the crescent being located in the upper lip, and the lower half of the crescent being located in the lower lip, of the most anterior portion of the calcarine cortex. Working with a chimpanzee, Spence and Fulton [6] extirpated the entire left occipital lobe and at a later date resected part of the right occipital lobe. Subsequently it was observed that the chimpanzee had vision in only the periphery of the left homonymous field. When the brain was finally examined it was seen that only the anterior portion of the right area striata was intact. These investigators concluded that their experiments supported the view that the anterior portion of the area striata around the calcarine fissure is the cortical terminus for peripheral vision.

Kronfeld [7] and Soriano [8] reviewed the cases reported in the literature. The etiology was varied. The best anatomic contributions were derived from the subjects with cerebral injury. In dealing with cases of tumor of the brain one cannot disregard the possibility of edema and other distant reactive phenomena as causal factors in disturbance in the field of vision, so that no exact anatomic information may be available. On the other hand, a good deal of pathologic physiology may be learned from tumor material. For example, when a tumor grows in or near the area occupied by the optic radiation, there results usually progressive alteration of perception in the contralateral field of vision; the change in the function of vision is gradual, although usually fluctuating. A study of this gradual change may reveal knowledge which could not be ascertained by other methods.

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3. Poliak, S.: The Main Afferent Fibre Systems of the Cerebral Cortex in Primates, Berkeley, Calif., University of California Press, 1932, vol. 2, p. 370.

4. Pfeiffer, R. A.: Myelogenetische Untersuchungen ueber den zentralen Abschnitt der Sehleitung, in Foerster, O., and Wilmanns, K.: Monographien aus dem Gesamtgebiete der Neurologie und Psychiatrie, Berlin, Julius Springer, 1925.

5. Traquair, H. M.: An Introduction to Clinical Perimetry, St. Louis, C. V. Mosby Company, 1927, p. 211.

6. Spence, K. W., and Fulton, J. F.: The Effects of Occipital Lobectomy on Vision in the Chimpanzee, Brain **59**:35, 1936.

7. Kronfeld, T.: The Temporal Half Moon, Tr. Am. Ophth. Soc. **30**:431, 1932.

8. Soriano, F. J.: Semiología de las hemianopsias homónimas, Semana méd. **1**:1005, 1931.

The exact incidence of occurrence of peripheral temporal crescentic defect is difficult to determine, because the visual fields could not always be determined perimetrically. Of one hundred patients with verified tumor implicating the optic radiations for whom the fields of vision were charted, ten showed defects of the temporal crescentic variety. The types of unpaired defects found in the peripheral temporal field of vision are illustrated and discussed in the following case reports.

REPORT OF CASES

HEMICRESCENTIC DEFECTS

CASE 1.—S. F., a boy aged 12, complained of occasional attacks of headache and nausea for six weeks prior to admission to the hospital; for twelve days he experienced twitchings of the right side of the body.

Examination revealed faciobrachial paresis on the right, with a concomitant sign of involvement of the pyramidal tract. The fundi were normal. Central visual acuity was 20/20 in each eye. Repeated studies showed a well defined hemi-

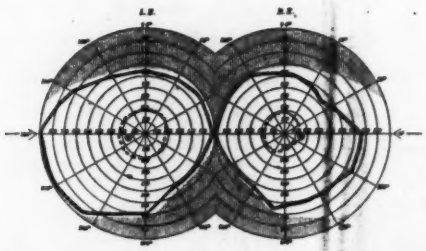

Fig. 2 (case 1).—Visual acuity was 20/20 in each eye. The fundi were normal. A crescentic defect in the upper and, to a lesser extent, in the lower right temporal field was revealed on examination with test objects 4 and 2 mm. in size.

All the observations of the visual fields in the studies reported were made with the Ferree-Rand perimeter. The test objects, the size of which is indicated in each case, were in motion during the examination. Daylight illumination was used. In the charts of the visual field the solid lines represent the border of the field for white objects, and the broken lines the border of the field for colored objects.

crescentic defect in the periphery of the upper and, to some extent, of the lower temporal quadrant of the right field of vision. The field for color was concentrically contracted in the right eye. The spinal fluid pressure was 120 mm. of water. Nine days after the patient's admission an encephalogram revealed normal ventricular and subarachnoid systems. Five days later a tumor (spongioblastoma multiforme) was removed at operation from the left paracentral lobule.

Comment.—In this case there was no evidence from the appearance of the fundus oculi, encephalogram and lumbar puncture to indicate the presence of distant pressure phenomena or distortion of cerebral or optic nerve structures to account for the hemicrescentic and the cres-

centic defect in the peripheral temporal field of vision. Since the pathologic signs were those of a malignant infiltrating tumor, it is possible that the defect of the field was the first manifestation that a part of the optic radiation conducting the fibers for the crescent was implicated by tumor tissue or by reaction around the neoplasm.

CASE 2.—A. F., a school teacher and housewife aged 48, complained that for four and one half years before admission to the hospital she experienced auditory hallucinations (heard her own voice but could not describe the words); for four years she had spells during which she imagined she saw objects; for two years she had disturbances of memory and attacks of petit mal.

Examination showed obesity, irregular pupils which reacted poorly to light and in accommodation, blurring of the margins of the optic disks and paresis of the supranuclear part of the facial nerve on the right. Though usually cheerful and somewhat facetious, she was irritable on occasions. She had slight difficulty in naming common objects.

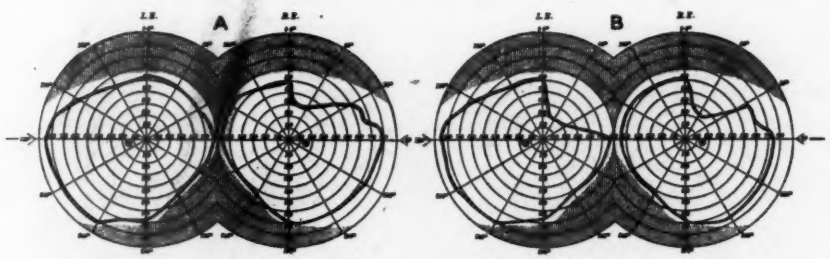

Fig. 3 (case 2).—There were complex hallucinations in the right field of vision. Central visual acuity was 20/20 in each eye. The fundus of each eye showed blurring of the edges of the disk. On examination with a test object 1 mm. in size a hemicrescentic defect was found in the right upper peripheral temporal field of vision (*A*), which when it became larger resulted in a homonymous field defect (*B*).

Four days after admission she became cooperative. A perimetric examination of the field, on July 6, 1935, consistently showed a hemicrescentic defect in the periphery of the right upper temporal field of vision (fig. 3*A*). Central visual acuity was 20/20 in each eye. Two days later she complained that she saw figures which appeared to be close to the eye in the right field of vision. On the same afternoon repeated visual studies indicated homonymous superior quadrantanopia in the right field, which was incomplete and incongruent (fig. 3*B*).

The patient refused operation. Three months later papilledema with hemorrhages developed bilaterally. At that time she was too uncooperative for studies of the visual fields to be made. Operation disclosed a huge meningioma located deep in the left sylvian fissure.

Comment.—In this case the unpaired hemicrescentic scotoma was a forerunner of incomplete homonymous superior quadrantanopia in the right field. It appears that as soon as the temporal defect in the right eye had reached a certain size the paired nasal quadrant in the other eye

became similarly affected. Interesting was the complex visual hallucination in the right field of vision which the patient experienced just before the perimetric examination.

CASE 3.[9]—J. K., a man aged 44, entered the hospital with the chief complaint of "bandlike headache." Four years before admission he had an attack of unconsciousness. Four and a half months prior to admission he suffered from headache and tremor of both hands. Two weeks later he began to vomit. Three weeks before entering the hospital he became unsteady in his gait and had weakness in the right arm and face; his vision became blurred, and the headaches felt more severe—"like a band around the left temple." During the last two weeks he had diplopia, polydipsia and drowsiness.

Examination revealed hemiparesis and diminished sensibility on the left side of the body and signs of involvement of the pyramidal tract bilaterally. All the ocular movements, including those of convergence, were slightly impaired. Both external rectus muscles were paretic. The fundi revealed papilledema, hemorrhages and exudates. The patient was drowsy and somewhat euphoric. The spinal fluid was under an initial pressure of 340 mm. of water.

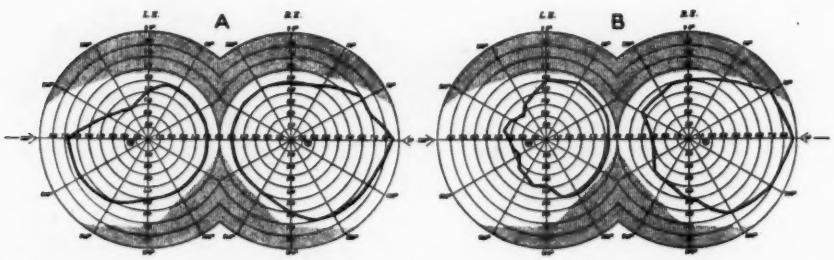

Fig. 4 (case 3).—There was blurring of vision. Central visual acuity was 20/50 in each eye. The fundus of each eye showed papilledema. There was a crescentic defect in the left peripheral temporal field of vision, which was most pronounced in the upper crescent (*A*). Ten days later, the field defect progressed to incomplete homonymous hemianopia, involving the binocular paracentral fields (*B*). Examination was made with a test object 4 mm. in size.

On March 18, 1932, the day following admission to the hospital, perimetric tests indicated a well defined hemicrescent in the left upper, and a narrow hemicrescent in the left lower, peripheral temporal field of vision (fig. 4 *A*). Central visual acuity in each eye was 20/50.

Three days later a ventriculogram taken after trephining in the left occipital region indicated what was interpreted to be a neoplasm compressing the right

9. The visual fields and observations at autopsy in this case were previously described by Globus and Silverstone (Diagnostic Value of Defects in the Visual Fields and Other Ocular Disturbances, Arch. Ophth. **14**:325-386 [Sept.] 1935). In connection with this case they called attention to the fact that in homonymous lateral anopias "the defect on the side opposite the lesion was almost always greater than that on the same side. . . An attempt to correlate these findings with the extent of the lesion found on postmortem examination did not yield reliable substantiating observations."

lateral ventricle from the midline and from below. Under observation partial ophthalmoplegia developed on the right (with ptosis of the upper lid, paresis of the superior, internal and external rectus muscles and dilation and fixation of the pupil). Craniotomy was postponed because of a preexisting infection of the scalp (a furuncle).

Perimetric examination, repeated ten days after the first test, on March 28, showed a total crescentic defect in the periphery of the left temporal field and a narrow defect in the periphery of the right nasal field of vision, so that the picture was one of incomplete asymmetrical homonymous hemianopia in the left field (fig. 4*B*). The infection of the scalp did not clear, and the patient died three weeks later from complicating erysipelas.

Autopsy discosed a large cystic tumor (spongioblastoma multiforme), which seemed to have originated in the region of the right hippocampal gyrus. It infiltrated the inferior half of the right temporal lobe and the anterior portion of the occipital lobe. The corpus callosum and the ventricular system were displaced to the left.

Comment.—In this case the earliest defect in the field of vision was closely similar to the type found in the two previous cases, yet the

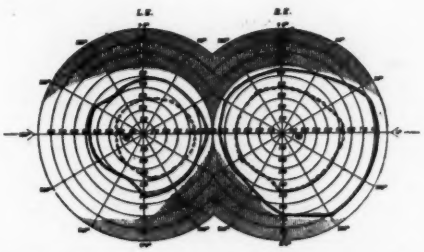

Fig. 5 (case 4).—There were crude visual hallucinations and blurred vision. Central visual acuity was 15/30 in each eye. The fundus of each eye showed papilledema. On examination with a test object 4 mm. in size a total crescentic defect for form and color was found in the left peripheral temporal field of vision.

location of the neoplasm was different in each case. Another feature was the incongruity of the defects in the fields of vision, the defect being more marked in the temporal than in the homonymous nasal field of vision.

PERIPHERAL TEMPORAL TOTAL CRESCENTIC SCOTOMA

Case 4.—J. S., a man aged 43, was readmitted to the Mount Sinai Hospital because of headache. After he first entered the hospital, ten months previously, he had a partial pneumonectomy and partial excision of the wall of the chest and the diaphragm for carcinoma of the right lung with invasion of the wall of the chest and the diaphragm. Six weeks prior to the second admission he had severe headache in the occipital and the right frontal regions and blurring of vision. Occasionally he saw streaks of light with his eyes closed. Distinct changes in personality were noted by his relatives.

Examination revealed bilateral early papilledema and a crescentic defect for both form and color in the left peripheral temporal field of vision (fig. 5). Central visual acuity of each eye was 15/30. There was paresis of the left central portion of the face and abdominal reflexes were absent bilaterally. Under observation he became drowsy, and the pulse rate dropped to 52 beats per minute. Lumbar puncture showed a pressure of 180 mm. of water, a total protein content of 175 mg. per hundred cubic centimeters and 4 cells per cubic millimeter. Exploratory craniotomy on the right gave negative results. The patient fared poorly and died two days later. Autopsy revealed edema of the right hemisphere, with displacement of the ventricular system to the left. On the ventral and mesial surface of the anterior portion of the occipital lobe and the posterior portion of the temporal lobe was a tumor measuring 3 cm. in the lateral diameter. The surrounding area was gray and soft. The tumor nodule had metastasized from a carcinomatous focus in the bronchus. There were no other metastases to the brain.

Comment.—In this case the defect in the field of vision illustrates the typical total crescentic defect in the peripheral temporal field.

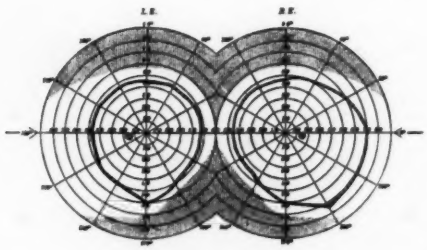

Fig. 6 (case 5).—Central visual acuity was 10/15 in the right eye and 10/30 in the left. The fundus of each eye showed blurring of the margins of the disk. On examination with test objects 4 and 2 mm. in size a total crescentic defect was found in the left peripheral temporal field of vision.

Although the neoplasm was well localized, there was too much surrounding edema to enable one to draw any definite conclusions as to the location of the fibers conducting vision from the periphery of the nasal retina through the optic radiation. The lesion implicated at least the lower portion of the optic radiation.

CASE 5.—B. O., a man aged 40, gave a history that nine weeks before admission to the hospital he felt a shock "like electricity" running through his left lower limb. Associated with this he experienced dizziness and headache on the right. Three weeks later the left upper and lower extremities felt "asleep" and weak. The appetite became poor, and he lost weight.

Examination showed diminished sensation in the left extremities, which was of the cortical sensory type. There was slight paresis of the supranuclear part of the facial nerve on the left. The abdominal reflexes were absent, while the deep reflexes were increased on the left. The optic disks were slightly blurred. Central visual acuity was 10/15 in the right eye and 10/30 in the left. By perimetric examination a total crescentic depression in the left temporal field

of vision was found. On the following day the examination was repeated and the crescentic defect found. Systemic examination gave negative results.

Exploratory craniotomy over the right parietal lobe revealed no evidence of a neoplasm. The patient died on the tenth postoperative day. Autopsy revealed one metastatic carcinoma nodule located in the right parietal lobe. The tumor, 2.5 by 3 by 3 cm. in size, was 0.5 cm. below the lateral and mesial borders of the right parietal lobe; it extended from a point 0.5 cm. in front of the right parieto-occipital fissure anteriorly to a point 1 cm. in front of the splenium of the corpus callosum. The tumor was clearly demarcated from the rest of the brain. There were a few small hemorrhagic areas in the periphery, while on the lower edge there was some softening.

Comment.—This scotoma is another example of crescentic defect in the temporal field. The major lesion in this instance bordered the upper rim of the radiation. Detailed histologic studies of the optic fibers and the occipital cortex were not available.

CASE 6.—A. F., a man aged 49, entered the hospital with the history that one month prior to admission he saw spots before the eyes. One week later he had stiffness of the muscles of the neck, and while driving an automobile he felt "shaky" because he could not judge distances between his and other automobiles. For two weeks prior to admission he noted progressive failure in vision, the sensation being "as though there were a cloud over the eyes." He walked unsteadily, and on many occasions he bumped into objects and missed his way. He feared that he might fall through the floor because he was not sure of his gait. There was clumsiness of finer movements of the hands. He complained of headache in the frontal region and sticking pains in both eyes, of dizziness and of noises like "wheel grinding" in the ears. His relatives noted that his memory became impaired. He became mentally sluggish and emotionally unstable.

Examination revealed a palpable mass of glands in the left supraclavicular fossa, slight clumsiness of the left hand and increased reflexes on the left side of the body. Occasionally he made errors in the position sense of the left fingers and toes, and there was some dystereognosis in the left hand.

The fundi on admission revealed early papilledema; later this became advanced, and exudates and hemorrhages appeared. A few hemorrhages were noted to be present near the maculae. Mentally the patient was confused, dull and poorly oriented and had a defect in regard to memory and general information. A biopsy of the cervical lymphatic glands in the left supraclavicular fossa showed that the enlargement was due to follicular lymphoblastoma. The patient was not sufficiently mentally alert for adequate studies of his fields of vision to be made until thirteen days after admission, on April 3, 1936. At that time he was cooperative, and the studies revealed an unpaired total crescentic defect in the most peripheral portion of the left temporal field of vision (fig. 7*A*). The peripheral nasal field of the right eye was studied carefully by asking the patient to focus on a point 30 degrees along the horizontal meridian located to the right (temporally) of the central point of fixation, the absolute field of vision thus being exposed.

Perimetric tests for color showed a contracted temporal field in the left eye and a generally contracted field in the right eye. Central visual acuity was 20/100 in the right eye and 20/40 in the left. Examination of the central fields

by the stereocampimeter showed an enlarged blindspot and a relative scotoma in the temporal half of the central field of vision of the left eye.

Eight days later a ventriculogram demonstrated a mass in the right hemisphere. At operation on the same day a highly malignant type of sarcoma the size of a plum was removed from the posterior part of the right parietal lobe and the anterior part of the right occipital lobe. The tumor, which was located deep in the brain substance, was encapsulated and contained a loculated cyst.

Four days after craniotomy there was complete homonymous hemianopia in the left field. This soon began to recede, and after two weeks, on May 2, a large zone of central vision returned (fig. 7 *B*).

Comment.—This case illustrates that the crescentic defect found in the peripheral temporal field of vision of one eye could not be detected in the corresponding nasal field of the other eye even after the absolute field was studied. The factor of obstruction of the field by the body of the nose, which bounds the relative field of vision, was eliminated by having the patient rotate the eye being tested temporally in the horizontal meridian. It was interesting to note that when the hemianopia began to recede central vision was the first to reappear.

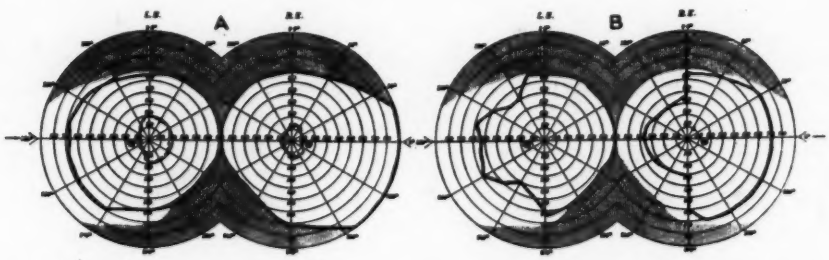

Fig. 7 (case 6).—There were crude visual hallucinations, inability to gage distances and cloudy vision. Central visual acuity was 20/100 in the right eye and 20/40 in the left. The fundus of each eye showed papilledema, with exudates and hemorrhages. There was a total crescentic defect in the left peripheral temporal field of vision (*A*). After the removal of the neoplasm from the right parieto-occipital lobe, there was complete homonymous hemianopia in the left field, which later regressed from the center toward the periphery (*B*). Examination was made with a test object 4 mm. in size.

PERIPHERAL TEMPORAL ABSOLUTE SCOTOMA AND RELATIVE CRESCENTIC SCOTOMA

Case 7.—F. C., a 52 year old woman, was admitted to the hospital complaining of headache of three weeks' duration. Two and a half weeks before admission she forgot how to write and had attacks of dizziness and weakness. During the week before admission vomiting developed; she became somewhat confused and had slight difficulty in understanding spoken and written language.

Examination revealed a slight increase in the muscular tone and in the reflexes on the right side of the body. There were minimal paresis in the right upper limb, mild hemihypalgesia on the right and bilateral papilledema with exudates

and hemorrhages. On gross examination a slight defect was seen in the right temporal field of vision. The patient had total alexia and agraphia. Spontaneous speech was hesitant and showed several minor errors. She was unable to concentrate. She appeared bewildered and depressed. The spinal fluid was slightly xanthochromic and under a pressure of 160 mm. of water; it contained 69 lymphocytes per cubic millimeter, and its total protein content was 84 mg. per hundred cubic centimeters of fluid.

On the day after the patient entered the hospital, on Aug. 19, 1934, perimetric examination revealed a crescent of absolute blindness in the right peripheral temporal field of vision and, mesially adjacent, a crescent of indistinct or relative blindness (fig. 8*A*). Because the patient became fatigued easily, the fields for color were not tested. Central visual acuity was 20/20 in the right eye and 20/50 in the left. Two days later the perimetric tests showed a slightly larger absolute

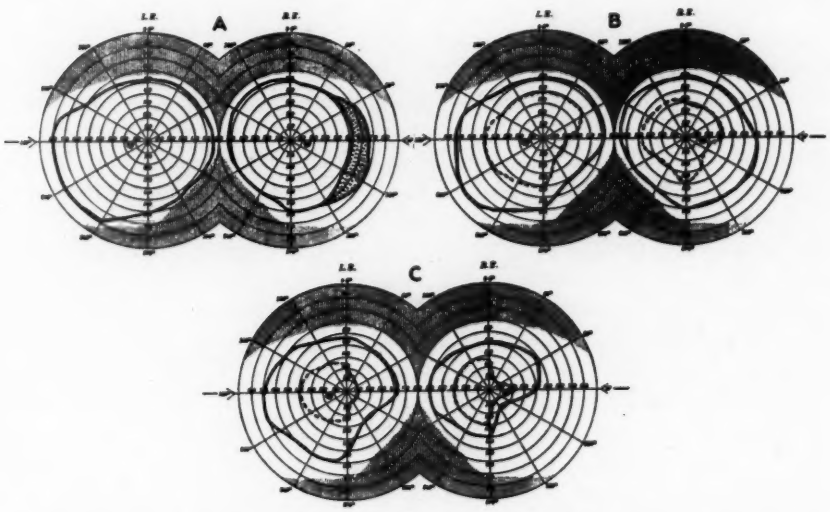

Fig. 8 (case 7).—There were alexia and agraphia. Central visual acuity was 20/20 in the right eye and 20/50 in the left. The fundus of each eye showed papilledema with hemorrhages. There were a total absolute and a relative crescentic scotoma in the right peripheral temporal field of vision, as shown in *A*. (The stippled area represents the crescent of indistinct vision.) Note that the progression of the hemianopia from the periphery toward the center was preceded by defects in the field for color, as shown in *B* and *C*. Examination was made with a test object 4 mm. in size.

crescentic defect, a small contraction in the left inferior peripheral nasal field and homonymous inferior incomplete quadrantanopia in the right field for color (fig. 8*B*).

On August 25, seven days after admission to the hospital, a ventriculogram taken after trephining in the right occipital region demonstrated a mass in the region of the left posterior horn. On the day after the studies with air were made, perimetric examination showed incomplete homonymous hemianopia in the right field, with a tendency for the inferior quadrants to be more defective (fig. 8*C*). Associated with this there was a homonymous hemiachromatopia in the right field, in which central vision was preserved.

On the following morning craniotomy revealed an infiltrating glioma (spongioblastoma polare) in the posterior and inferior portions of the left parietal lobe, located 5 cm. below the surface of the brain.

Comment.—In this instance there was a crescentic defect which was split in the longitudinal meridian, causing an absolute peripheral crescent and, mesial to this, a crescent of transient or "relative" blindness. Equally interesting was the progression of the unpaired crescentic defect to homonymous incomplete hemianopia and quadrantanopia. It should be noted that the defects in the field for color preceded those in the field for form, indicating that more "central fibers" were implicated, as well as the "peripheral fibers," and that the hemianopia had advanced from the periphery.

Case 8.—E. G., a 52 year old man, entered the hospital complaining of difficulties in memory and in calculation. Three years before admission he sustained an injury to the head, at which time amber-colored fluid escaped from the left ear.

Five months prior to admission he experienced numbness of the fingers in the right hand. Three months later he had a fainting spell, and since then he had felt "pressure" in the back of the head. In addition he complained of impairment of memory, had difficulty in recalling names of persons and numbers and made mistakes in calculations and in reading printed words. On occasions he jumbled his words in conversation.

Examination revealed normal spontaneous speech. However, careful analysis showed that the patient omitted and transposed letters in writing, mixed syllables in reading and made errors in calculation, especially in subtraction and division. Previously he had been an excellent mathematician. He also had echolalia. Lumbar puncture showed a clear fluid under a pressure of 210 mm. of water. A roentgenogram of the skull revealed a calcified pineal body in the normal position.

On Nov. 23, 1934, two days after admission, perimetric tests disclosed a total crescentic defect in the right peripheral temporal field of vision (fig. 9 *A*).

The field for color in the right eye showed a tendency to incomplete homonymous inferior quadrantanopia. Reexaminations on the following day and on two subsequent occasions showed an absolute crescentic defect in the field for form and that for color in the right peripheral temporal field of vision; mesial to this defect was a crescentic relative scotoma. The fields in the left eye were normal (fig. 9 *B*). Central visual acuity, uncorrected, was 10/50 in the right eye and 10/20 in the left.

The fundi were myopic and showed a slight myopic conus in each eye. Nine days after admission an encephalogram indicated that a mass was present in the region of the left temporal lobe. At exploration a tumor the size of a hickory nut was removed from the posterior portion of the junction of the left temporal and parietal lobes; it was at a depth of 5 cm. Autopsy later showed the cavity which was left by the removed tumor to be located deep in the midportion of the left temporal lobe, lateral to the convexity of the inferior temporal horn of the lateral ventricle.

Comment.—This is an example in which two crescents were demonstrable in one peripheral temporal field of vision, the more peripheral scotoma being absolute and the more central being relative. Equally interesting were the findings in the fields for color, which also showed the temporal crescent defect in the right eye. Also of note was the slight incipient defect in the field for form found in the inferior nasal quadrant of the left eye. The homonymous inferior quadrantanopia in the right field for color seemed to be confirmatory evidence for the foregoing defects in the visual field. The quadrantic defects were not found on subsequent examinations. The recession of the defect in the field may have been due to fluctuations in the course of the disease, which are often observed in cases of tumor of the brain.

CASE 9.—A. P., a 27 year old art student, entered the hospital with the history that two years before admission she imagined she saw flashes of light. Two weeks before admission she vomited and suffered headache in the frontal region and dizziness. A day before admission there were double vision and numbness in the right arm.

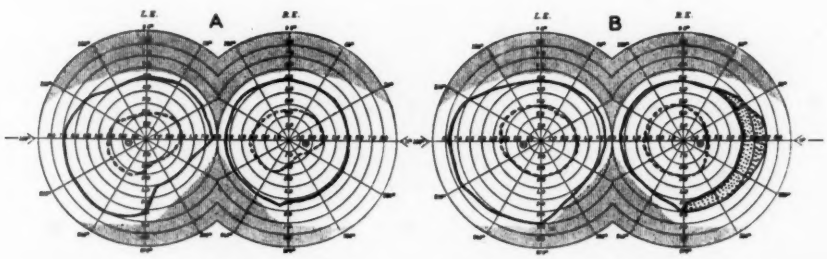

Fig. 9 (case 8).—There were dyslexia and dysgraphia. Central visual acuity was 10/50 in the right eye and 10/20 in the left. The fundus of each eye showed myopia. The fields (*A*) were similar to those in figure 8 *B*. Two days later, on November 25, the defect regressed, and the crescent of indistinct vision (the stippled area) appeared (*B*) as in figure 8 *A*. Examination was made with a test object 4 mm. in size.

On examination the positive findings were: hemihypalgesia and facial weakness on the right, blurring of the margins of the optic disks, mental dulness, inability to concentrate and euphoria. The fields of vision were not plotted; grossly there was no hemianopia. A roentgenogram of the skull showed increased convolutional markings and a calcified pineal gland which was displaced to the right. A ventriculogram followed by craniotomy revealed a glioma the size of a walnut in the left parietal lobe. The patient made an uneventful recovery.

Five months later she was readmitted to the hospital because of headache and vomiting. At this examination there were dystereognosis and impaired position sense in the right extremities. There was slight aphasia, with dyslexia and dysgraphia. The fundi were normal. The cranium was reexplored, and more tumor tissue was removed from the same region.

One year after the first operation the patient was readmitted a third time because of the same symptoms. It was reported that she had a visual hallucina-

tion, the details of which could not be ascertained. Physical examination showed more marked sensory changes and aphasia than on previous admissions. She was extremely cooperative in all kinds of performance tests. Gestalt tests showed her to be a "visual" subject. Although she had slight difficulty in reading and writing, she was able to draw objects at sight with great skill and in detail. Plotted visual fields showed an absolute crescentic scotoma and mesial to this a relative crescentic scotoma, in the right peripheral temporal field of vision. There was homonymous hemianopia for color in the right field, which was more marked for green than for red. Central visual acuity was 20/40 in the right eye and 20/30 in the left. The fundi were normal. The patient was operated on a third time, and at this operation the glioma was found at the juncture of the left parietal, temporal and occipital lobes. Six weeks after the last operation complete homonymous hemianopia was found by confrontation tests.

Comment.—Despite the fact that the visual fields were plotted some time after the original operation, this case may be used as another example of absolute peripheral temporal crescentic scotoma and relative crescentic scotoma. The incomplete homonymous hemianopia for color,

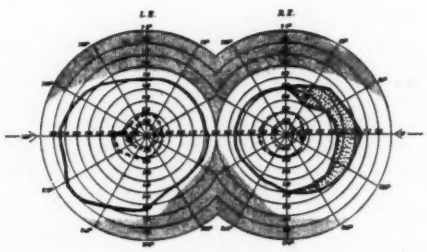

Fig. 10 (case 9).—There were crude and complex visual hallucinations, with dyslexia and dysgraphia. Central visual acuity was 20/40 in the right eye and 20/30 in the left. The fundus of each eye showed slight blurring of the margins of the optic disk. There were a total crescentic scotoma in the right peripheral temporal field of vision and, mesial to this, a crescentic relative scotoma (the stippled area). There was incomplete homonymous hemianopia for color, which was greater for green (the field bordered by the dash line) than for red (the field bordered by the dotted line).

which was more marked for green than for red, indicated that function of many fibers in the dorsal and ventral halves of the optic radiation had been impaired.

Case 10.—J. W., a 42 year old man, was admitted to the hospital with the history that two weeks prior to admission he was found on the bathroom floor in an unconscious and rigid state. A few days later there developed stiffness of the neck and inability to read or to find words or to concentrate properly. One day before admission he had headache and nausea.

Examination revealed a pulse rate of 64 beats per minute. There was mild sensory aphasia, with dyslexia, dysgraphia and dysnomia. Paresis of the right-central portion of the face and slight signs of involvement of the pyramidal tract on the right were evident. Mentally, the patient was irritable; he appeared

depressed and somewhat bewildered. His memory for immediate events was markedly impaired. The fundi showed slight blurring of the nasal margins, and the veins appeared engorged. Perimetric tests revealed homonymous hemianopia for color, with preservation of central vision, an irregularly crescentic area of absolute blindness in the right peripheral temporal field of vision and, centrally adjacent to this, an irregularly crescentic zone of partial blindness (fig. 11). There was a tendency for the defects to be greatest in the inferior halves of the crescents. In the other eye, along with a slight defect in the inferior nasal quadrant, there appeared to be incomplete incongruent homonymous inferior quadrantanopia. Central visual acuity was 20/20 in each eye.

Four days after admission exploratory craniotomy was performed. The patient died one month later. At autopsy a tumor (spongioblastoma multiforme) was observed in the ventral half of the temporal lobe, involving the posterior portions of the optic radiation. Surrounding this area there was edema.

Comment.—This case demonstrates the relative crescentic defect, and since such a zone of indistinct vision has been found in three previous cases, it begins to assume significance. It would appear that the

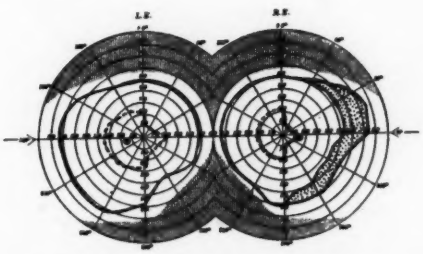

Fig. 11 (case 10).—There were dyslexia and dysgraphia. Central visual acuity was 20/20 in each eye. The fundus of each eye showed slight blurring of the nasal margins of the disk. On examination with a test object 4 mm. in size a relative cresentic scotoma (the stippled area) in the right peripheral temporal field of vision and incipient homonymous hemianopia for color in the right field, with preservation of central vision, were found.

temporal crescent may be split longitudinally, and this suggests the possibility that the fibers in the radiations have a lamellar distribution and are arranged in definite order. The incongruity of the hemianopia in the field for color and the disproportion between the field for form and that for color indicated an active process in which the optic fibers were irregularly affected.

GENERAL COMMENT

In the foregoing cases of neoplasm implicating the optic radiations it has been shown that crescentic or hemicrescentic areas of blindness may be found in the peripheral temporal field of vision of one eye. In corroboration of this observation were the concomitant defects found in the field for color (cases 4, 6 and 8) and the crescentic relative scotoma

which were found to be mesially contiguous to the peripheral temporal crescentic absolute scotoma (cases 7, 8, 9 and 10). Cases illustrating a sparing of hemicrescentic and crescentic areas in the peripheral zone of the temporal half of the field with homonymous hemianopia have been described by Harris,[10] Behr,[11] Delepine [12] and Traquair.[5]

These clinical observations, as well as anatomic studies of experimental and pathologic specimens, gave ample proof that the most peripheral portions of the temporal field of vision are unpaired and unilaterally represented in the opposite optic cortex. The exact route of the fibers from the retina to the lateral geniculate body was traced by Brouwer and Zeeman,[1] and that from the lateral geniculate ganglion to the calcarine cortex by Poliak.[3] Prior to 1932 the location and distribution of the optic fibers in the radiation were moot points. Pfeiffer [13] concluded that the upper rim of the lateral portion of the optic radiations contained the fibers for central vision and that the lower rim contained the fibers for peripheral vision. From this one may infer that the fibers for vision in the temporal crescent traverse the ventral part of the optic radiation.

Duke-Elder [14] stated that these fibers have a special localization in the central optic pathways, in the anterior part of the medullary optic lamina, being finally projected in the anterior part of the optic cortex. Some authors are of the opinion that the fibers are in the mesial portion of the ventral rim. From our clinical studies it appears that the fibers for peripheral temporal vision may be contiguous with their respectively paired quadrants of paracentral and central vision, the arrangement being lamellar (fig. 1). It is probable that the fibers for monocular vision (in the temporal crescent) do not intermingle with those subserving binocular vision.

The optic radiation is composed of individual bundles, each having its definite subcortical origin, its course and its cortical termination. Our observations are in agreement with those made in the anatomic studies of Poliak.[3] He summarized that the most dorsal bundles of the optic radiation transmit vision from the opposite upper peripheral

10. Harris, W.: Hemianopia with Especial Reference to Its Transient Varieties, Brain **20**:308, 1897.

11. Behr, C.: Die homonymen Hemianopsien mit einsietigem Gesichtsfelddefekt, im "rein temporalen halbmondförmigen Bezirk des binokularen Gesichtsfeldes," Klin. Monatsbl. f. Augenh. **56**:161, 1916.

12. Delepine, S.: Case of Right Lateral Homonymous Hemianopsia: Arteriosclerotic Softening of the Left Cuneus, Tr. Path. Soc. London, **41**:2016, 1890.

13. Pfeiffer, R. A., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 1, p. 426.

14. Duke-Elder, S.: Text Book of Ophthalmology, St. Louis, C. V. Mosby, 1934, vol. 1, p. 273.

nasal retina, which in the field of vision corresponds to the lower peripheral temporal hemicrescent, while the most ventral bundles transmit vision from the contralateral lower peripheral nasal retina, which receives impulses from the upper peripheral temporal hemicrescent in the field of vision (fig. 1).

DIRECTION IN WHICH HOMONYMOUS ANOPIAS DEVELOP

Irrespective of the location of the lesion in the optic radiation, homonymous lateral anopias have been observed to begin in the periphery with a crescentic or hemicrescentic scotoma in the temporal field of vision (cases 2, 3, 7 and 9). Subsequently the blindness progressed toward the center and frequently did not involve central vision. During the process of regression the reverse phenomena occurred (cases 6 and 8)—the range of central vision gradually increased to include the paracentral, and finally the peripheral, field of vision. The foregoing statement is illustrated by the following case report:

CASE 11.—A 34 year old school teacher was admitted in stupor to a neighboring hospital. Examination showed a stiff neck, fever, glycosuria and 30 lymphocytes per cubic millimeter of spinal fluid. Three days later she was transferred to the Mount Sinai Hospital, and on admission mixed aphasia and weakness of the right-central part of the face were found. Under observation, defects in the right field of vision appeared and disappeared, as shown in the charts (fig. 12).

The diagnosis rested between neoplasm of the left temporal lobe and encephalitis. An encephalogram showed no abnormality. The patient was discharged from the hospital in a much improved condition. The nature and location of the lesion were not verified.

This patient showed the temporal crescentic defect at the onset and end of homonymous hemianopia.

Although Peter,[15] in his book on perimetry, stated that anopias may begin in the center of the field and develop peripherally, such observations have not frequently been made in cases of tumors of the temporal lobe. Globus and Silverstone[16] noted that there was no essential difference in the type of defects of the visual field in cases of tumor which involved the mesial or lateral portions of the temporal lobe. In most instances of homonymous lateral anopias due to lesions of the radiation the defect does not include central vision. If the fibers for central, paracentral and peripheral vision have a definite position in the supragenículate pathways, why should not a lesion destroying, for example, the fibers for central or paracentral vision produce

15. Peter, L. C.: The Principles and Practice of Perimetry, Philadelphia, Lea & Febiger, 1931.

16. Globus, J. H., and Silverstone, S. M.: Diagnostic Value of Defects in the Visual Fields and Other Ocular Disturbances, Arch. Ophth. **14**:325 (Sept.) 1935.

homonymous anopia starting in the central or paracentral zones and developing toward the periphery rather than starting from the periphery and developing toward the center? Why is it that central vision is the last to disappear and the first to recover, irrespective of which fibers of the optic radiation are destroyed first?

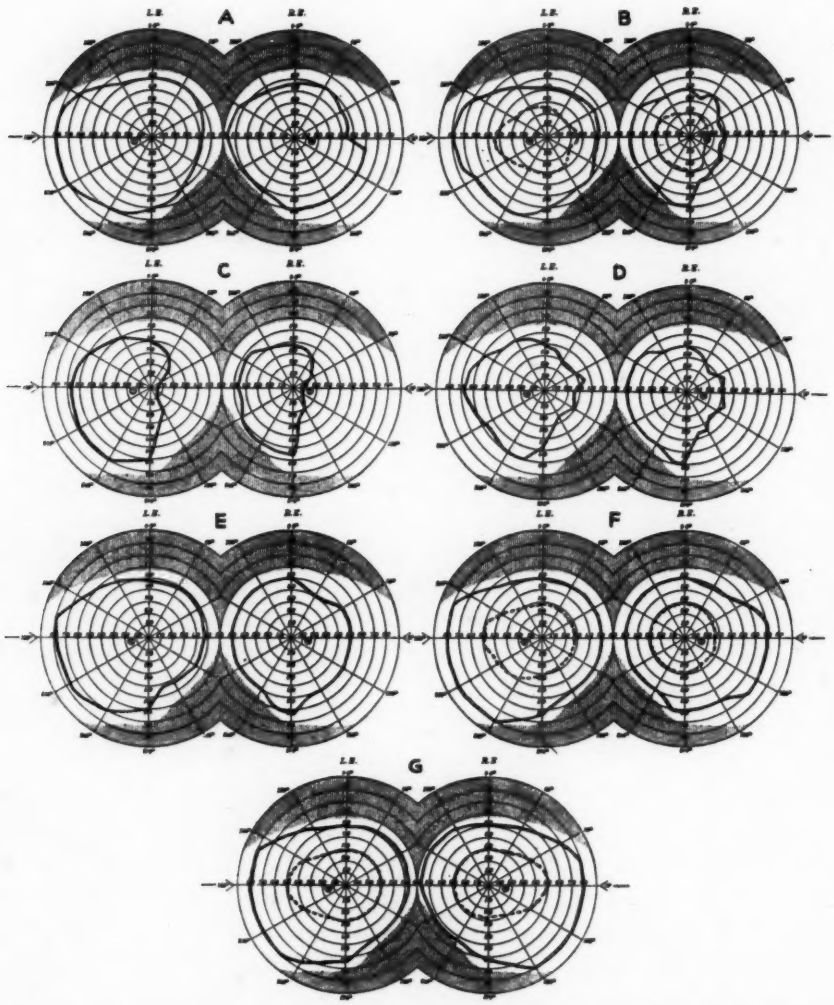

Fig. 12 (case 11).—There were alexia and agraphia. Central visual acuity was 20/20 in each eye. The fundi were normal. The charts of the visual fields show the development and regression of homonymous hemianopia in the right field. The letters indicate the fields on the following dates: *A*, Jan. 4, 1934; *B*, January 7; *C*, January 9; *D*, January 13; *E*, January 26; *F*, February 20, and *G*, March 1. Note that the peripheral temporal crescent in the field for form and in that for color was the first to be affected and the last to return to normal. Examination was made with a test object 4 mm. in size.

The answer to these questions may be derived from the following observations and theories. The macular and paracentral fields constitute the binocular field of vision. The periphery of the temporal field of vision is monocular (not paired in the homonymous nasal field) and unilaterally represented. Since the eyes operate as a unit, it is probable that structures subserving vision for the paired central and paracentral fields and the unpaired peripheral temporal fields are closely related in their anatomic and physiologic features.

Traquair [5] has shown that sensitivity to a white object, or visual acuity in the relative field of vision, was highest in the center and gradually decreased toward the periphery. Visual acuity varies with the density of the retinal elements (rods and cones). The number of rods and cones per unit area is greatest in the central region and least in the periphery. Brouwer and Zeeman [1] traced the optic fibers from the retina and found a progressive increase in the area occupied by the fibers for central vision as they approached the lateral geniculate body. The unpaired peripheral fibers occupied a narrow rim in the ventral portion of the external geniculate ganglion (fig. 1). Pfeiffer [13] traced the fibers farther along the geniculocalcarine pathway through the external sagittal stratum of the optic radiation. He stated that in the dorsal part of the stratum sagittale externum coursed the macular fibers. Henschen [17] concluded that the bundle of fibers for peripheral vision occupied a part which was 1 cm. high in the ventral portion of the external rim; the greatest area was occupied by the fibers for macular vision. Poliak [3] inferred that about half of the area of the entire optic radiation represents central vision. Holmes,[18] from his studies of gunshot wounds in the occipital lobe, proposed that the serial concentric zones in the retina from the macula outward to the periphery seem to be represented in the same order from behind forward in the occipital cortex, and that the macular area is relatively large, while the peripheral area is relatively small. The relative sizes of the areas covered by the fibers for central, paracentral, and peripheral vision are illustrated in figure 1.

Thus it is seen that central vision is transmitted by fibers greater in number than those which represent the paracentral or the peripheral field of vision. The area occupied by fibers for macular vision in the geniculocalcarine pathways gradually increases as they approach the occipital cortex. It is probable that the change in the number of optic fibers and in the area covered by these in the retinogeniculocalcarine

17. Henschen, S. E., quoted by Pfeiffer,[13] p. 436.

18. Holmes, G.: Disturbances of Vision by Cerebral Lesions, Brit. J. Ophth. 2:353, 1918.

pathway is proportional to the gradual change in the visual acuity at their corresponding points in the field of vision. In other words, a direct relationship exists between function and structure.

Physiologically, an interesting and important feature in the intensity of the defects is the relation of the loss in the field for color to that in the field for white (cases 1, 4, 6, 7, 8, 9 and 10). In partial impairment of function, vision for color is first diminished. Only when the impairment has reached a certain degree is vision for white demonstrably reduced. In a diseased area either a large number of nerve fibers may be slightly affected, the remainder being healthy or seriously damaged, or, on the other hand, there may be a small percentage of severely damaged nerve elements fairly evenly distributed, all the others being healthy or a few being slightly diseased. In both instances visual acuity for white and for color is reduced, but in the first instance the loss of vision for color is excessive. The relative numbers and the distribution of the healthy, partly damaged and severely damaged nerve structures vary according to the nature and location of the lesion. The nature of the intensity of the corresponding defect in the field may safely be regarded always as the resultant of the aforementioned factors. The relation between the uniformity of the defect and that of the lesion is complicated by the varying value of the visual acuity between the center and periphery of the field and by the question whether by the same degree of damage functional impairment is as easily produced in one set of fibers as in another.

With these theories in mind it should become clear why a lesion, especially a tumor, located anywhere near or in the optic radiation will be manifested by homonymous anopia which begins in the periphery and gradually develops toward the center. Because the fibers for peripheral vision are fewest, cover the least area and, proportionally, conduct vision of lowest acuity, a tumor that directly or indirectly destroys the function of a few of these fibers will promptly reduce the visual acuity in the corresponding field. On the other hand, the tumor may affect primarily or simultaneously a greater portion of the fibers for central or paracentral vision and yet, because the optic fibers occupy a larger area in the radiation, are greater in number and conduct vision of increasing acuity, the visual acuity in the corresponding field will decrease in the periphery first, for the reasons already given. When the lesion affects the fibers of either the dorsal or the ventral half of the radiation, the defect in the visual field will start as a hemicrescentic scotoma in the periphery of the temporal field and develop into homonymous quadrantic anopia (case 2); if the lesion progresses to involve the other half of the radiation (as in case 3—the dorsal half

after the ventral half), homonymous anopia will occur, and if the edema or the tumor affects one half of the radiation more than the other (as in case 7), irregular quadrantic defects in the field for form and hemianopia in the field for color will be found. When the disease process affects the central fibers of both halves of the geniculocalcarine pathway, there will follow homonymous anopia, at first for color (case 10) and then for form (case 11).

The same explanation applies to regressing hemianopia in which the pathologico-physiologic condition is reversed. Although these deductions may explain preservation of central vision in hemianopia caused by a lesion implicating the optic radiation, they do not confirm or disprove that macular vision may be bilaterally represented.

Objections to the foregoing theories may be advanced from examples of hemianopia in which peripheral temporal vision was spared. In most of those cases the lesions were unverified and the localization was based on clinical grounds. In the cases in which the disease process was verified (usually cases of arteriosclerosis or gunshot wounds), the location was in the cortex of the occipital lobe, where the physiologic and structural components for vision are different from those of the optic radiations in the temporal lobe.

Riddoch [19] studied recovery from hemianopia following gunshot wounds, and he found that recovery occurred from the periphery to the center of the field. The remaining defect in the central or paracentral field was loss of appreciation of movement; this zone was surrounded by a zone of loss of recognition of objects. In some instances he found regression beginning with a temporal crescent. These observations are in direct contrast to those made in patients with tumor of the brain and require explanation. The location of the lesions in Riddoch's cases were estimated from the points of entrance and exit of the missile and, in some cases, by operation. The extent of the lesion could not be adequately determined. In some of Riddoch's patients the defects discovered seemed to be migrating from one field to another, implying that they were progressive or regressive bilateral lesions of the optic pathways. All the lesions that he found were in the occipital lobe. Furthermore, it must be remembered that in cases of gunshot wounds the damage to the tissues in question is sudden and massive. The lesion is at its maximum in the first few days. When the surrounding edema disappears there is a residual defect in the optic pathways or the occipital cortex. Thus, at first there will be complete homonymous hemianopia, which later decreases to a focal scotoma, the

19. Riddoch, G.: Dissociation of Visual Perception Due to Occipital Injuries, with Especial Reference to Appreciation of Movement, Brain **40**:15, 1917.

nature of the latter depending on the number of fibers or cells destroyed and the location of the scar in the geniculocalcarine pathway. In cases of tumor of the brain the sequence of events is different. The disease process is expanding and usually continual. The anopias in cases of tumor of the temporal or parietal lobe are produced more likely by reactive phenomena than by direct neoplastic infiltration of the broad but very thin band of optic fibers which constitute the optic radiation. The recovery in the field of vision which is found at some interval after extirpation of the tumor is due to decrease of pressure, edema or other effects which impeded the function of the optic fibers. Discrete residual scotoma may be observed if a part of the band of radiation is destroyed during the operation. Unfortunately, most neoplasms which involve the radiation directly and produce cerebral symptoms are large, so that in removal of the tumor most of the radiation may be destroyed, and there will be no recovery of visual function. Even if only a part of the optic fibers are extirpated with the neoplasm, by the time the partially damaged fibers would have recovered function (and a great number of fibers would be impaired if the tumor were in that location), the neoplasm would probably recur. Another reason why residual scotomas are not commonly recorded in cases of tumor of the brain postoperatively is that even if the patient lived long enough he might not be available for observation.

Thus we saw that the earliest sign of involvement of the optic radiation was a crescentic defect in the peripheral portion of the opposite temporal field of vision. Strauss [20] emphasized the necessity for studying cases of tumor of the brain early in the course of the disease. Such a practice will undoubtedly permit the temporal crescentic scotoma to be more commonly recognized than it has been. Indeed, this defect may even be found to be the most frequent type of distortion in the fields of vision of patients with tumor of the brain.

SUMMARY

Cases are reported in which a lesion of the optic radiation produced an unpaired crescentic or hemicrescentic defect in the periphery of the temporal field of vision. In several instances there was a concomitant defect in the field for color.

In four cases an unpaired crescentic relative scotoma was found mesial to the peripheral crescentic absolute scotoma. This observation suggested the probability that the fibers in the optic radiations have a lamellar arrangement.

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20. Strauss, I.: The Initial Symptoms and Early Diagnosis of Tumor of the Brain, Bull. New York Acad. Med. **12**:467 (Aug.) 1936.

The most dorsal bundles of the optic radiation transmit vision from the opposite upper peripheral nasal retina, while the most ventral bundles transmit vision from the contralateral lower peripheral nasal retina.

Irrespective of the location of the lesion in the radiation, homonymous anopias usually begin in the periphery and advance toward the center, frequently sparing central vision. The regression of the anopsias occurs in the reverse manner. These phenomena are explained on a functional and structural basis.

An unpaired peripheral temporal scotoma indicates an early defect in the optic radiation. Such a scotoma has localizing value in the early diagnosis of tumor of the brain.

ASTIGMATIC DIALS IN REFINED REFRACTION

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I believe, as John Green [1] stated in 1866, that astigmatic dials, properly used, are the most dependable practical devices yet discovered for measuring the amount and axis of astigmatism. Failure is due to faults of equipment or technic. Lancaster [2] was too modest to say this in 1915 when he presented his paper on subjective tests for astigmatism, but if one had read between the lines or taken the trouble to test himself or his patients by this method, one would appreciate why this master of the art of refined refraction anticipated a rational response, namely, the general adoption of his dial or other astigmatic dials by ophthalmologists.

Twenty years later one finds earnest, conscientious, painstaking refractionists struggling without the satisfaction which astigmatic dials would afford or, perhaps, using only one dial—the clock-face or sunburst dial—with disappointing results. What is the reason? The chief reason is that most textbooks containing chapters on refraction and even textbooks on refraction deal with the technic for the use of astigmatic dials in so inadequate a fashion that beginners, after trying to follow the incomplete directions, are not satisfied with the results and give up this method to seek some more reliable method for determining the presence, amount and axis of astigmatism—and, incidentally, there is none.

Few American authors and fewer European authors have written about the use of astigmatic dials. Directions have been brief and have usually dealt with the use of the clock-face dial, which, if used without a supplementary dial, is inadequate, for the following reasons:

1. The lines 30 degrees apart do not admit fine discrimination of axes.

2. If the examiner attempts to neutralize the astigmatism (to determine the amount of astigmatism), by using one dial, he finds that before the lines in the principal meridians (for example, the vertical and horizontal lines) become equally black, the patient states that the oblique lines and not the vertical (or horizontal) lines appear blackest.

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1. Green, John: Annual Report of the Netherlands Ophthalmic Hospital, Utrecht, 1866.

2. Lancaster, W. B.: Subjective Tests for Astigmatism, Especially Astigmatic Charts, Tr. Am. Acad. Ophth., 1915, p. 167.

It appears, therefore, that a supplementary dial of two principal meridians, as suggested by Verhoeff [3] in 1899 must be used in conjunction with the clock-face (or sunburst) dial to obtain the best results. My experience with Green's, Verhoeff's, Thorington's and Friedenwald's dial and with other dials has resulted in the conclusion that Lancaster's 50 cm. dial with lines 20 cm. long, about 2 minutes wide and 10 degrees apart, is the most refined. Regarding a supplementary dial with which to determine the amount of astigmatism, I think that equal accuracy can be obtained with single crossed lines of different lengths and widths; but, as Lancaster pointed out, since the patient's vision must be fogged to obtain accurate results, the lines must be wider than 1 minute.

Green's [1] original lines, as well as Wallace's,[4] were designed "to subtend an angle of one minute for the purpose of making recognition of the lines in any one meridian the standard of perfect vision in that meridian." After working with numerous dials between 1866 and 1878, Green concluded that the lines on some of his original dials were too narrow for the best results at six meters.[5] He found that better results were obtained by using wider lines. This was confirmed by Lancaster, clinically and photographically. Nevertheless, many writers have failed to appreciate the importance of this point. Thus, Thorington [6] stated that in most charts the lines subtend an angle much greater than the Snellen standard for the distance at which they are used, and "in this way the true delicacy of the test for small errors or amounts of astigmatism is sacrificed." Friedenwald's charts fall into the same error. Charts with narrow lines like Friedenwald's may give better results than Lancaster's for a small percentage of patients, viz., those few patients who combine unusually good powers of observation with small errors of refraction. It should be distinctly understood that what I am maintaining is that wider lines give better results for a larger percentage of patients.

The simplicity and economy of astigmatic dials should make them the universal choice of refractionists, especially those who, from choice or necessity, work without a cycloplegic. Helmholtz [7] used small figures made up of numerous radiating lines to illustrate the general phenomena

3. Verhoeff, F. H.: Two New Astigmatic Charts, Ophth. Rec. **8**:541, 1899.

4. Wallace, J.: An Improved Astigmatic Chart, Univ. Med. Mag. **2**:13, 1889-1890.

5. Green, J.: On a New System of Tests for the Detection and Measurement of Astigmatism, Tr. Am. Ophth. Soc. **4-5**:131, 1867-1868.

6. Thorington, J.: Refraction and How to Refract, ed. 3, Philadelphia, P. Blakiston's Son & Co., 1904, p. 138.

7. Helmholtz, H. L. F.: Handbuch der physiologischen Optik, Leipzig, L. Voss, 1909, pl. 2, fig. 5.

of astigmatic vision, and Donders [8] in 1860 or earlier used a star-shaped figure made up of fine radiating lines as a practical test for astigmatism. Snellen adopted a dial similar to Green's "B" dial, but reduced the number of lines from sixty (3 degrees apart) to eighteen (10 degrees apart) and used only the upper half—the astigmatic fan—regarding which Green [9] had said that it "has the single advantage of falling within the limits of the Octavo page of his test types."

That the presence and axis of astigmatism can be demonstrated by dials is therefore an old story, but pitfalls due to lack of reliable technic must have been many, else, in my opinion, no refraction would be concluded today without resorting to the dials.

Evidence tending to prove the superiority of the Lancaster dials and his technic for obtaining the best clinical results with them were presented so lucidly and convincingly by Lancaster [2] in 1915 that one has but to read the article, procure the charts and proceed to better results in refraction. True, the original charts are not on the market and must be made by gluing black velvet ribbon on wedding stock paper, but Bausch and Lomb [10] manufactured a set of greater durability. These were square boxes about 2 inches (5 cm.) in depth, with the lines cut out on metal fronts, but the cost of these seems to have been more than most refractionists wished to pay.

Another drawback to the popularity of the dials is their size. They require more wall space than many wish to give, or can give, to dials. Lancaster explained that if the dial is small, "a 10° interval brings the lines pretty close together"; in his larger dial his intention was to avoid this and at the same time have the lines 10 degrees apart, this arrangement being, I believe, superior to that in which the lines are 15 or 30 degrees apart.

The aforementioned difficulties, viz., the trouble (and skill) required to make the dials, the expense of the manufactured set and the economy of wall space, are all legitimate deterrents to the adoption of the dials, especially by the beginner, unless, of course, he has used them while in training and already considers them indispensable. When Lancaster's attention was called to this, he offered to aid me in my effort to modify his original dials. After months of trial we produced the dials here offered, which have given results almost as satisfactory as the original Lancaster dials. These dials are now available at nominal cost and require about the same wall space as Verhoeff's dials.

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8. Donders, F. C.: Beiträge zur Kenntnis der Refractions- und Accommodationsanomalien, Arch. f. Ophth. (pt. 1) **7**:155, 1860.

9. Green, J.: Test-Diagrams for the Detection and Measurement of Astigmatism, Tr. Am. Ophth. Soc. **2**:467, 1878.

10. Bausch and Lomb issued a booklet giving instructions by Lancaster in the use of the charts.

SUGGESTED EXPERIMENTS WITH LANCASTER-REGAN ASTIGMATIC DIALS TO SHOW THE PRINCIPLE INVOLVED

Those who are not skilled in the use of astigmatic dials (and this includes some who think they are but who are not) will find the following experiments a valuable means of clarifying the principles involved.

How to Illuminate the Dials.—The charts are hung in a good light of at least 15 to 25 foot-candles, the whole wall where they are hung

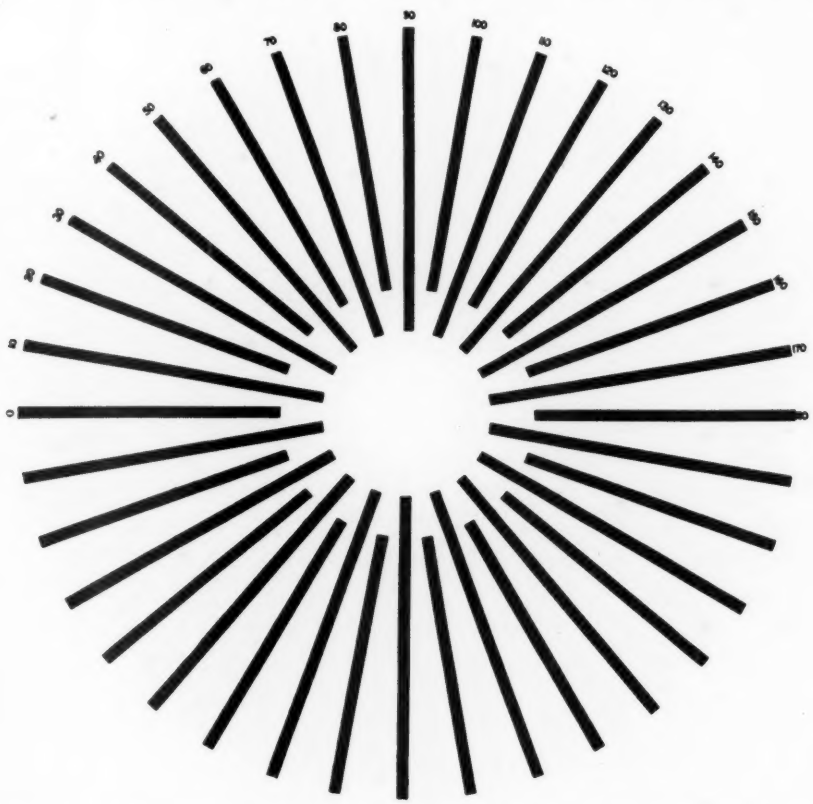

Fig. 1.—Astigmatic dial 1, with thirty-six radiating lines at intervals of 10 degrees.

being illuminated with flood-lights so that the space around the dials will be almost as bright as the dials themselves. A dark background should be avoided, for two reasons: First, it is desirable to have a volume of light entering the pupil such as will produce a size of pupil equal to that found under average working conditions—neither the large pupil produced by dim illumination nor the small pupil produced by overbright illumination is desirable. The larger pupil introduces into the problem the factor of refractive errors outside the visual zone of the

cornea. These are often substantial. The small pupil reduces the size of circles of diffusion in a way similar to a stenopaic disk, although, of course, to a less degree, thus obscuring the effects of astigmatism. Second, the retina responds more sensitively (as was shown by Cobb and Moss [11]) when the surrounding field is such that there is not a marked contrast in brightness between the chart and the surrounding field.

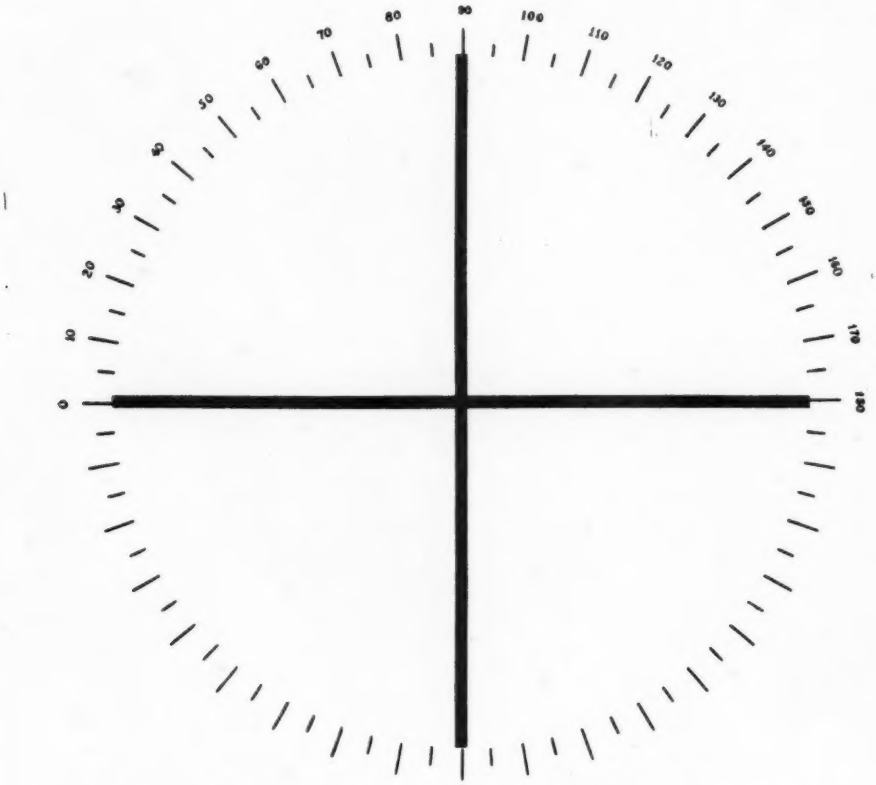

Fig. 2.—Astigmatic dial 2, with a cross adjustable at any angle.

The following experiments may be tried on one's own eyes if one has approximately normal vision—vision of 6/8 or better. If not, an intelligent patient may be used, preferably one with an accommodation of at least 4 D. and a pupil about 4 mm. in diameter.

One should start with the eye corrected to approximate emmetropia.

Experiments on Fogging.—A trial frame is placed comfortably over the glasses ordinarily worn. The eye not tested is occluded with

11. Cobb, P. W.: The Effect on Foveal Vision of Bright Surroundings, J. Exper. Psychol. **1**:540, 1916. Cobb, P. W., and Moss, F. K.: The Effect of Dark Surroundings on Vision, J. Franklin Inst. **206**:827, 1928.

an opaque disk. The smallest letters possible are read with the chart at 6 meters.

Experiment 1: A + 0.25, a + 0.50, a + 0.75 and a + 1.00 sphere are placed in the trial frame in succession; the smallest letters possible are read in each case, and a note is made of these. This will be referred to as "fogging."

This should show a definite reduction in vision for each addition of a plus sphere. If no definite reduction occurs with a + 0.50 sphere, this shows that there is some uncorrected hypermetropia. An emmetropic eye with a + 0.50 sphere reads about two lines less on a Green-Ewing chart.[12]

Experiment 2: The same procedure as that in experiment 1 is carried out, but a — 0.25, a — 0.50, a — 0.75 and a — 1.00 sphere are used.

This should show no definite reduction in vision if the subject has good active accommodation.

Experiment 3: Experiment 1 is repeated, a series of opaque disks with apertures of different sizes ranging from 5 to 2 mm. being used to reduce the size of the pupil. It should be found that the effect of fogging is markedly less with a small pupil.

Experiment 4: Experiment 1 is carried out on the other eye, and the findings are recorded. If the corrections worn by the eyes are well balanced, the reduction in vision will be about the same in each eye.

Experiment 5: Experiment 1 is carried out on both eyes simultaneously. In the average case the reduction in vision will be markedly less than when one eye is tested, showing that the subject relaxes his accommodation more when both eyes are tested together.

Experiment 6: Experiment 1 is repeated stronger lenses (up to + 4 D.) being used, with the test object at 40 cm. (seen by the emmetropic eye with a + 2.50 D. lens).

Experiment 2 is repeated, with the test object at the same distance (40 cm.), with stronger concave lenses (up to whatever power is needed to take up all the accommodation and produce some reduction in vision).

Experiments in Astigmatism.—Experiment 7: This experiment is like experiment 1, except that instead of observing the test letters the subject observes the astigmatic dial 1 (the dial with thirty-six radiating lines at intervals of 10 degrees).

If the subject sees all the lines alike—equally distinct or, when vision is fogged, equally blurred—either he has no marked regular

12. The great advantage of the Green-Ewing chart is that the test letters are graduated by geometric progression, making the ratio of difference in size the same with each successive line. For example, the letters on the 6/10 line are 25 per cent larger than the letters on the next line; these, in turn, are 25 per cent larger than the letters on the next line, and so on for the whole scale.

astigmatism which is uncorrected by his glasses or, if he has some uncorrected astigmatism, he is not a keen enough observer to detect it.

Experiment 8: (*a*) A plus sphere is placed in the trial frame over one eye (the other eye being occluded with a blank disk); a sphere which will fog the vision to 6/10 or 6/8 should be chosen.

(*b*) A —1.00 cylinder, axis 90 is added, and the appearance of the astigmatic dial is observed and recorded. The line at 90 degrees and one or possibly two lines on each side of it are much darker, more distinct and better focused than any of the others.

(*c*) The axis of the minus cylinder is changed from 90 degrees to various other degrees, and note is taken of how clearly lines in the chart correspond.

(*d*) Experiment 8 *b* and *c* is repeated, —0.75, —0.50, —0.25 and —0.12 cylinders being used.

Experiment 9: Experiment 8 is repeated with less fogging and without fogging.

Experiment 10: Experiment 8 is repeated, —0.25, —0.50 and —0.75 spheres being used. It is noted that with the conditions as in experiment 8, i. e., with vision fogged, the chart shows the clear lines in the same axis as the minus cylinder. But under the conditions of experiment 10 there may be no marked difference in the radiating lines with a cylinder which is easily detected when the vision is fogged; then, as the minus sphere is increased, the time comes when the clearest lines are at right angles to the axis of the cylinder. With a little practice and by selecting the proper combinations of a plus sphere and a minus cylinder at different axes, the subject can make different axes come out clear at will; e. g., with a minus cylinder, axis 45 he can make the lines at 45 degrees clear, or by changing his accommodation he can bring out the lines at 135 degrees.

This shows how worthless the astigmatic charts are for finding the axis of astigmatism unless the accommodation is cared for by proper fogging.

It should be noted also that with a small amount of astigmatism no difference in the radiating lines is detected if the fogging is too great. The ideal fogging is produced by the sphere which fully corrects the hypermetropia plus another sphere which equals the amount of the astigmatism; e. g., if the true refraction is + 1.00 sphere ⊃ + 0.75 cylinder, axis 80, the best fogging to show the astigmatism by the dials is obtained with a + 1.75 sphere.

Hence, when the amount of astigmatism has been determined in a given case, it is often wise to corroborate the axis by placing in the trial frame the sphere which equals the stronger axis in hypermetropia,

and that which equals the weaker axis in myopia, and determine the axis of astigmatism again on dial 1.

Experiment 11: In a series of cases of astigmatism, either natural or produced by suitable spherocylindric lenses, the axis is first determined by the method just described and then the amount is measured by use of dial 2, the dial with the cross adjustable at any angle.

The cross is set at the angle determined by the test with dial 1. One line will be clearer than the other. This is the axis of the plus cylinder required. The other line will be less clear. This is the axis for a minus cylinder to correct the astigmatism. Minus cylinders are tried at this axis until the one is found which makes the two lines alike (not the one which makes this line as distinct as possible, for this would overcorrect the astigmatism). When the correct power is found the two lines of the cross will be equally distinct. (If a strong minus cylinder is required, the plus sphere must be made stronger to maintain the fogging.)

The first estimate of the amount of astigmatism, or, in other words, of the strength of the cylinder required to correct it and make both meridians equal, has been made. One is now ready to measure the spherical error more accurately, after which the amount of astigmatism will be verified and a still more accurate measurement made.

The fogging is reduced until the maximum vision is found. A power such that the addition of a $+0.50$ sphere reduces the vision from 6/5 to 6/8 is selected. The effect of a $+0.25$ cylinder and then that of a $+0.12$ cylinder is tried, with the axis first in line with the correcting cylinder and then at right angles to it. Unless the hypermetropia of the subject is not fully corrected, he should promptly and consistently tell which way the cylinder is held. The cylinder held at either axis will blur the axis on the chart, thus making the opposite axis the more distinct one.

SOME DIFFICULTIES ENCOUNTERED

When vision is adequately fogged, a subject, after viewing dial 1 for several minutes, often insists that all thirty-six lines look alike. If a -0.50 cylinder, axis 90 is held before the eye, he may respond that the lines are still alike. If the cylinder axis 180 is held before the eye, the subject may respond that the horizontal lines are blackest. His attention should be directed to dial 2, with the lines vertical and horizontal, and he can easily see that the horizontal line is blacker. Minus cylinders, axis 90 are inserted until both lines appear equally black; one should not be surprised to find that frequently astigmatism of 0.50 D. or more exists. Of course, when the amount of astigmatism is determined in this manner great care must be exercised in determining the axis, and in such cases I suggest testing the patient again with dial 1.

Rarely, the subject will see no difference in the lines with a —0.50 cylinder in any position, and the response is that all the lines on dial 1 look double or shadowed. If so, his vision may be overfogged—an error which may occur when the astigmatism is slight. Some persons will select lines in two directions—not at right angles. In these cases the astigmatism is slight and somewhat irregular. The subject is using a special part of the focal pencil which nowhere, in his case, brings the light to a perfect point focus. He chooses the place which gives him the best results in seeing.

ANOTHER DIFFICULTY

The subject selects the blackest lines on dial 1 and after the cross-dial (dial 2) is set, minus cylinders up to, say, —0.75 are inserted. At this point the response may be: "Both lines appear double, but equally black." The most frequent cause of this is fogging. One must make sure of this by holding a minus sphere before the eye. This usually removes this doubling. If not, the axis is tried again with dial 1. There are two ways of selecting the axis at this point: The sphere is reduced sufficiently to give vision of 6/6; then the minus cylinder is removed from the frame, and the subject's attention is directed to dial 1. The center of the group of black lines may now appear to be 5 or 10 degrees on either side of that originally selected by the subject. The cross-dial is set at the new axis. The spherical power is increased to refog vision; the —0.75 cylinder is inserted, and one proceeds toward accurate measurement of the amount of astigmatism. Another method is to reduce the sphere until the patient has vision of 6/5 and rotate the cylinder while the patient views letters on the 6/8 or 6/6 line. The axis to choose is the one which gives the best focus of these letters.

The astigmatic dials mentioned in this article are manufactured by the E. B. Meyrowitz Surgical Instruments Co., Inc., 520 Fifth Avenue, New York.

p_H AND BUFFERS IN RELATION TO OPHTHALMOLOGY

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The determination of p_H [1] has proved of great value in the perfection of many processes in various important industries. The usefulness of this test in the canning of fruits and vegetables has paralleled its application in the baking, tanning and sugar industries. Manufacturers of pharmaceutic products, those engaged in the dairy industry and many others employ p_H for one purpose or another in the betterment of their products.

In the basic sciences, such as bacteriology, biology and pathology, as well as in dentistry the use of p_H is well established. Thus it has been noted that there is a definite relationship in cow's milk between the p_H and the content per cubic centimeter of leukocytes and streptococci.[2] The importance of knowing the p_H of culture mediums and the rôle of p_H in bacterial growth is well known. Studies on the p_H of saliva in relation to the decay of teeth as well as in a number of physiologic and pathologic conditions have contributed much to the knowledge of physiology, bacteriolgy, immunology, pathology and other branches of medicine.

In this article, I have attempted to study p_H further as it relates to the problems and concerns of ophthalmology. Before results of these studies with p_H and also with buffers are stated, some consideration should be given to the method of approach and to the underlying principles behind the applications of p_H and buffers in ophthalmologic work.

The control of the p_H of a solution or ointment is of value, since it gives an accurate quantitative reading of the acid, alkaline or neutral reaction. The p_H bears no relation whatever to the osmotic pressure. Its determination is evolved around the theory of Arrhenius, wherein it is assumed that when an acid or an alkali is dissolved in water it breaks into ions (atoms) or groups of the same, which contain positive or negative charges of electricity. For example, water (H_2O) will be

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Read before the Section on Ophthalmology of the College of Physicians of Philadelphia, Jan. 21, 1937.

1. The p_H is the logarithm of the reciprocal of the hydrogen ion concentration. It may be defined as the power of 10 which indicates the fraction of a gram of hydrogen ions per liter of solution.

2. Baker, J. C., and Van Slyke, L. L.: A Method for the Preliminary Detection of Abnormal Milks, Bulletin 71, New York Agricultural Experiment Station, June 1919.

electrolyzed into H (+ ions) and OH (— ions). Determination of p_H is considered a more refined and delicate test than is a test with litmus.

A solution definitely shown to be acid or alkaline by determination of the p_H may sometimes give a neutral reaction to litmus. Titration gives the amount of acid or alkali in a solution. It does not indicate the intensity or the degree of the acid or alkaline content of the solution. A striking example of the information to be obtained from titration and p_H was given by LaMotte.[3] Three acids—hydrochloric (corrosive), acetic (used in vinegar), and boric (used in eye washes)—when taken in equally concentrated solutions in a tenth-normal titration bear a relation of acid intensity in the order of 15,000:200:1. These three acids, respectively, with the same value of tenth-normal titration show, however, different values for p_H—1,2.9 and 5.2, respectively.

By using determinations of p_H Nakashima[4] found the hydrogen ion concentration of the dark-adapted eye (retina) to be p_H 7.3, while in the illuminated eye it is p_H 7 or less.

The p_H scale as devised by Sorenson is similar to that of the metric system, wherein the division of p_H measurements is in multiples of 10. It is illustrated by figure 1, in which the apex of the two sides of a triangle at 7 represents the neutral point, or p_H 7. From this point, on one side of the formed angle centimeter divisions represent successive alkaline increase from p_H 7 to 14, while on the other side divisions from p_H 7 to 1 indicate definitely increasing acidity.

Each millimeter measurement on the scale in the illustration represents the fraction of p_H. Thus, p_H 4.5 is represented at 4.5 cm. on the scale; p_H 6 is 10 times as acid in reaction as p_H 7, p_H 5 is 100 times as acid as p_H 7, and so on, in multiples of 10 for the acid or alkaline side of the scale. The value p_H 7, or neutrality, is supposed to be represented by that of pure water.

METHODS OF OBTAINING MEASUREMENTS OF p_H

There are two methods of checking p_H, viz.:

1. The colorimetric test, which is a comparative color test. In this test indicators which are very sensitive to small ranges of p_H are used. A set of about a dozen would be sufficient for work in ophthalmology. Clark,[5] Lubs and others have prepared sets of these dyes which are commonly used. In this test the unknown solution, with the indicator

3. LaMotte, F. L.: The A B C of p_H (Hydrogen Ion) Control, ed. 10, Baltimore, Waverly Press, Inc., 1934, p. 21.

4. Nakashima, M., in Krause, A. C.: The Biochemistry of the Eye, Baltimore, Johns Hopkins Press, 1934, p. 85.

5. Clark, W. M.: The Determination of Hydrogen Ions, ed. 3, Baltimore, Williams & Wilkins Company, 1928, p. 411.

added, is compared with a series of stable buffered solutions of known p_H. When the color of the unknown and that of the known buffered indicator are exactly matched, the p_H of the unknown solution will be identical with that of the known control solution.

A modification of this colorimetric test is similar to the technic just described, except that papers saturated with indicators are used instead of tubes containing the buffer solutions used as indicators. A drop of the unknown solution is placed on the bibulous indicator paper, and this is compared with a color chart until the proper matching of color is noted. The p_H is then recorded. Another variation of this test is the

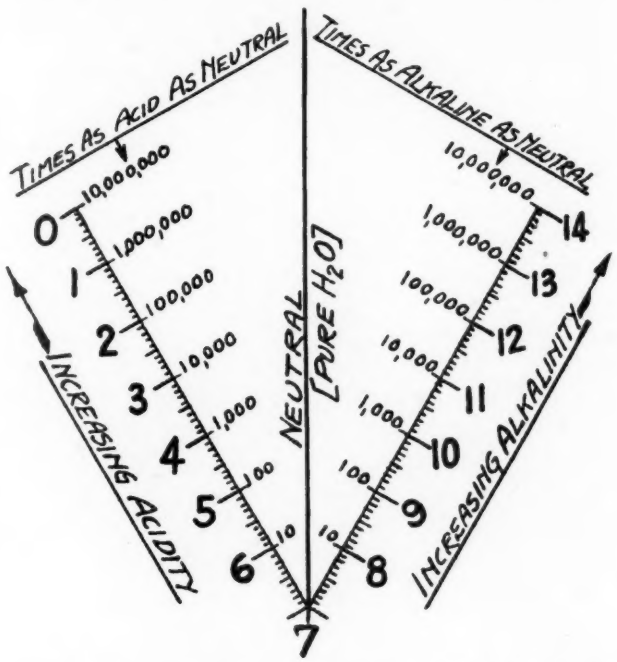

Fig. 1.—Diagram showing increase of acidity and alkalinity by logarithmic progression.

use of an indicator in the unknown solution. The resultant color is compared with the master color chart as already noted. The modifications just given are of value for the rough determination of p_H. The readings are obtained quickly, but small variations in p_H cannot be ascertained. Indicators and standard color charts are now available through a pharmaceutic concern.

2. The method I used for about a year is the electrometric or potentiometric test. Several concerns make the necessary apparatus. This method is accurate, and the technic is simple. It enables one to ascertain fractional differences in p_H.

The instrument I use contains a double stand (A) with an upper shelf holding three tubes for testing the p_H of alkaline solutions and a lower shelf holding a like number of tubes for the examination of the p_H of acid solutions. The middle tube of both sets contains a supersaturated solution of potassium chloride in distilled water. A buffer known to have a p_H of 7 is placed to the right of the solution of potassium chloride for examination of the alkaline solution to be tested. For testing an acid solution I used a buffer known to have a p_H of 5. The empty bottle to the left of the solution of potassium chloride on each shelf is used for the unknown solution. The buffer known to have a p_H of 7 was devised by Sorenson; the solution with a p_H of 5 was suggested by McIlvaine. When an acid solution with a p_H up to 7 is to be tested,

Fig. 2.—Potentiometer for the electrometric determination of p_H.

a pinch of quinhydrone powder is stirred into both the solution of unknown p_H and the buffer solution known to have a p_H of 5. Platinum terminals placed in both the solution of known p_H and the solution of unknown p_H are connected by leads to their respective posts (B) in the potentiometer (C).

U tubes (J) containing agar saturated with potassium chloride serve as conductors between the tube containing the solution of known p_H, the middle tube containing saturated solution of potassium chloride and the tube containing the solution the p_H of which is to be tested. In the testing of alkaline solutions no quinhydrone is used, and the terminals are antimony electrodes. The U tubes are used in the examination of alkaline solutions, as in the aforementioned procedure for the testing of acid solutions. Only one solution of unknown p_H, either acid or alkaline, can be tested at one time. The switch D opens the circuit, and

the p_H is quickly read on the dial (E). The aforementioned tubes are suitable when one has a fairly large amount of solution to work with—at least a few cubic centimeters of the unknown solution.

When only a small quantity of the solution of unknown p_H is available, as in the examination of tears, a Cullen tube (G) must be used. The Cullen tube[5] is held upright by the stand F. In the center of this tube is a removable platinum wire, to which, when it is moistened with distilled water and immersed in quinhydrone powder, the powder adheres. The Cullen tube is now ready for use in making a determination of p_H. The platinum wire with the holder is inserted into the pipet portion of the Cullen tube and the tears of unknown p_H can be sucked in by the slow withdrawal of the arm (H). There is a minimum of exposure of the tears to the air. Care must be taken that no quinhydrone drops from the platinum wire into the eye, for if this occurs the eye will for a short time become red and irritable. The test for p_H with the Cullen tube is simple and finds its greatest usefulness in cases in which only very small quantities of solution (1 or 2 drops) can be obtained. The Cullen tube with the solution of unknown p_H is then inserted into the supersaturated solution of potassium chloride. A U tube containing agar saturated with potassium chloride connects the solution of unknown p_H in the Cullen tube in the saturated potassium chloride solution with the buffer solution of known p_H 5, to which a pinch of quinhydrone is added. The leads of the Cullen tube and platinum terminal in the buffer solution of known p_H 5 are connected with the potentiometer. The current is switched on, and the p_H of the unknown drop or two is quickly indicated on the dial of the potentiometer, as in the examination of large quantities of unknown solution. Protein and salt "errors" as well as dichromatism, which commonly occur in the colorimetric test, are not a factor in potentiometric determination. Turbid mixtures and ointments can be easily examined by the electrometric method. In both the colorimetric and the potentiometric examination care should be taken that the temperature of the solution of unknown p_H is about room temperature. This is necessary because p_H is the amount of ionization of a mixture, and this ionization is increased with heat. The buffer with a p_H of 5 may be used for testing solutions the p_H of which ranges through the values for acid and up to values for alkali of about p_H 8. The buffer standard solution of p_H 7 is best used for alkaline solutions with a p_H of from 7 to 14.

BUFFER SOLUTIONS

A buffer solution is one the known p_H of which is not materially affected by a reasonable addition of weak acid or alkali, i. e., a solution which is resistant to alterations in its p_H. Any solution may be strongly

or weakly buffered. Buffer solutions are sometimes made by including an acid with the salt of that particular acid, such as boric acid and the borates, or citric acid and the citrates. LaMotte, Kenny and Reed,[6] as well as other authors of textbooks on p_H, have given a list of buffers of this type and others. A well known human buffer solution is blood. Human blood is buffered[7] in part by virtue of the presence within it of phosphates, carbonates and bicarbonates. MacLeod[8] quoted Van Slyke as stating that the normal range of the p_H of the blood is between 7.3 and 7.5. At p_H 7, coma occurs; at p_H 7.8, tetany sets in.

As has been mentioned previously, all the indicator solutions used in the colorimetric test are made with buffer solutions, and the solutions with p_H 5 and those with p_H 7 used in the potentiometric test here described are also buffered solutions. These solutions are reasonably stable and will retain their p_H for some time. A striking example of this stability is shown by a patient whom I treated. She used half of a 2 ounce (59 cm.) bottle of buffered eye wash the p_H of which was 7.6. She felt relieved and then put the medicine aside. When she returned after eleven months for refraction she asked whether the same medicine could be used again. I rechecked the p_H of the solution. It was 7.58.

Regardless of what ingredients are employed in making the buffer solution, great care must be used. The drugs should be weighed accurately, and the solution should be dated. The work should be entrusted to a careful technician or pharmacist. It is advisable, in making medicines for the eye, that the p_H of the buffer be clearly stated on the bottle in much the same way as is done by manufacturers selling nutrient mediums to bacteriologists. Only in this way can one check the p_H stability of preparations whenever desired.

TEARS

Hosford and Hicks[9] quoted Brown as showing tears to be sufficiently buffered to permit a dilution of from 1 to 15 with neutral distilled water without altering its p_H. I have been fortunate in being able to verify the findings of Brown. A patient with glaucoma whose tears had a p_H of 7.15 was the subject. Distilled water of p_H 6.8 was instilled drop by drop into the eye. After the instillation of each 2 drops of

6. LaMotte,, F. L.; Kenny, W. R., and Reed, A. B.: p_H and Its Practical Application, Baltimore, Williams & Wilkins Company, 1932, p. 20.

7. Remington, J.: Practice of Pharmacy, Philadelphia, J. B. Lippincott Company, 1936, p. 544.

8. MacLeod, J. J. R.: Physiology and Biochemistry in Modern Medicine, St. Louis, C. V. Mosby Company, 1930, p. 625.

9. Hosford, G. N., and Hicks, A. M.: Hydrogen Ion Concentration of Tears, Arch. Ophth. **13**:18 (Jan.) 1935.

water the p_H was determined. Not until the addition of the eighteenth drop of distilled water did the p_H fall to 7.13.

About fifty eyes were examined for the express purpose of finding the p_H of tears. The subjects were patients who suffered no particular ocular discomfort but who came on account of a refractive error. The p_H ranged from 5.2 to 8.35. The greatest number had a p_H between 7 and 7.4. Tears of an equal number of eyes of patients suffering from extra-ocular diseases such as acute and chronic catarrh, dacryocystitis, foreign body, trachoma and gonorrhea showed almost the same range of p_H, the value being in the neighborhood of 7 in the majority of cases. The hydrogen ion concentration was not typical for any disease examined. The tears of both eyes in any one patient in health or disease were not always of the same p_H, nor was the p_H the same for the same eye when the p_H of the tears was rechecked at a later date. Tears were examined at different times of the day.

I was again able to verify the findings of Hosford and Hicks [9] as to the wide range of the p_H of the tears of persons with gonorrhea. Even in cases of vaginal gonorrhea [10] the p_H varied between 4.4 and 7. This wide divergence in p_H might be, as was pointed out, the cause for the great resistance to cure which patients with the disease show, since the gonococcus can grow in mediums the p_H of which is not necessarily fixed within narrow limits. I was able to observe, in the study of the p_H of tears, only one case of gonorrheal ophthalmia at the very onset of the disease. The tears showed a p_H of 7.1 for the diseased eye and a p_H of 7 for the normal eye.

It is important to remember in examining the p_H of tears that an accurate test is obtained only when there is a natural flow, i. e., when the lacrimal apparatus is not artificially stimulated. The method which I have utilized when tears were not easily obtained consisted in instilling 1 or sometimes 2 drops of distilled water of known p_H into the lower cul-de-sac. A well is created by the patient gently pulling forward the lower lid. The patient is told to rotate the eye in all meridians. The tears are sucked into the pipet of the Cullen tube and are examined with the potentiometer. Whenever distilled water is used, the p_H of the water must be known to make sure that the tears and not the water is being examined. When in doubt a reexamination should at once be made with another drop or two of water to see whether the p_H varies. It is important to know the p_H of the distilled water used.

The use of lens paper to absorb tears and then examination by the colorimetric test is not as accurate a method. Indeed, I have immersed lens paper in distilled water and have found an increase in alkalinity

10. Nine patients from the clinic for patients with genito-urinary diseases of St. Christopher's Hospital for Children were examined.

of the water from p_H 6.5 to 7. It is important to bear in mind when examining tears for p_H that the patient has not been using medicaments in the eyes for some time, since prolonged use of ophthalmic medicines may alter the p_H of the tears.

DISTILLED WATER

In figure 1, p_H 7 was supposed to represent the p_H of neutral distilled water. However, such an assumption is not absolutely correct in all cases, since the p_H of distilled waters may differ. Gifford [11a] quoted Truog as giving 5.8 as the p_H of distilled water. I examined fifteen samples of distilled water obtained from an equal number of drug stores, and obtained the following readings for the p_H of the specimens by potentiometric tests, in the order obtained: 5.18, 4.88, 5.78, 5.9, 6.18, 6.2, 7.1, 7, 7.48, 6.1, 6.6, 5.9, 6.5 and 5.65. The fifteenth specimen was triple-distilled water in a sealed ampule. It had a p_H of 7.1 and when left exposed for ten minutes showed a p_H of 6.8. One reason for the variation in p_H of the distilled waters examined may have been that the makers of the water exposed it to the air in order that the beverage might be more palatable. Water absorbs 0.03 per cent of carbon dioxide from the air; this increases its acidity and reduces its p_H beyond the neutral range of p_H 7. It is possible that the difference in p_H of the various distilled waters may account for the wide variation between the values for hydrogen ion concentration of Gifford and Smith [11b] and those for the hydrogen ion concentration of a set of the same ophthalmic medicaments (table 1, columns 1 and 2) compounded at two reliable drug stores. Column 1 of table 1 gives the ophthalmic medicines compounded by druggist A and dispensed in cork-stoppered bottles. Druggist B, using the drugs from the same manufacturer, dispensed the mixtures in bottles with screw caps (column 2). The third column gives the p_H of the same medicaments that were prepared in buffer solutions and placed in cork-stoppered bottles. This was done to show the relative value of the two methods of dispensing ocular medications (in bottles with cork stoppers and in bottles with screw caps).

The composition of the average medicine bottle, I have learned, contains too little alkali to be of serious consequence in affecting the stability of the p_H of the medicament. All the bottles in each group were opened daily for ten minutes and then closed until the following day. The purpose of this was to simulate the amount of exposure when the contents were used during a two month period. This method is not above criticism, since it exaggerates the use to which the contents of the bottles are put. It may be argued that a 1 ounce bottle opened so much every

11. (*a*) Gifford, S. R.: Reaction of Buffer Solutions and of Ophthalmic Drugs, Arch. Ophth. **13**:81 (Jan.) 1935. (*b*) Gifford, S. R., and Smith, R. D.: Effect of Reaction on Ophthalmic Solutions, ibid. **9**:227 (Feb.) 1933.

day for use would not last two months. I have purposely made the second reading of the p_H after subjecting the medicaments to more severe use than would ordinarily be expected of them. The fact was, however, that all the bottles were subjected to the same treatment whether they held buffered or unbuffered medicines. This method served only as a comparative means of testing the stability.

In table 1, the p_H in ordinary type is the hydrogen ion content just after the medicine was made; the p_H in boldface type is the value at the end of two months, after the bottle was opened daily for ten minutes. It will be noted that the p_H of the same preparation differs

TABLE 1.—*p_H of Preparations Made with Distilled Water, Dispensed in Bottles with Cork Stoppers and in Bottles with Screw Caps, Compared with the p_H When a Buffer Solution and a Cork Stopper Are Used*

	p_H of Preparations Made with Distilled H_2O by Druggist A, in Bottles with a Cork Stopper		p_H of Preparations Made with Distilled H_2O by Druggist B, in Bottles with a Screw Cap		p_H of Preparations Made with Buffer Solution in Bottles with a Cork Stopper	
Drug Used, 1 Oz. Mixtures	First Reading	Reading After 2 Mo.	First Reading	Reading After 2 Mo.	First Reading	Reading After 2 Mo.
Atropine sulfate, 4 grains.....	5.75	**5.40 M**	6.20	**5.10**	7.55	**7.12**
Butyn, 2% solution.....	6.20	**5.25 P**	6.30	**5.75**	Not made	
Boric acid, saturated solution...	4.80	**4.90 M**	5.00	**5.18**	6.30	**6.30 M**
Boric acid-sodium borate solution	9.10	**9.00 M**	9.10	**9.08**	8.50	**8.45 P**
Cocaine, 2% solution.....	6.10	**4.15 M**	5.80	**5.60 M**	5.94	**4.08**
Physostigmine sulfate, 2 grains	6.15	**6.50 M**	6.90	**5.70**	7.50	**7.30 P**
Physostigmine sulfate, 1 grain..	6.10	**6.20 M**	6.20	**5.56 M**	7.50	**7.63 P**
Phenacaine hydrochloride, 1 grain	6.48	**5.08 M**	6.50	**5.40**	5.50	**5.90**
Homatropine hydrobromide, 1 grain	7.70	**5.73 M**	6.95	**8.19**	7.30	**7.70 P**
Metaphen, 1:10,000 dilution.....	8.95	**7.88 M**	8.87	**7.81**	7.58	**7.20**
Procaine hydrochloride, 4% solution	5.70	**6.50 M**	5.80	**6.12**	7.16	**6.18 P**
Ethylhydrocupreine hydrochloride	6.20	**5.95 P**	Not obtained		Not made	
Pilocarpine, 1% solution.....	5.20	**5.20 P**	5.94	**6.05**	7.28	**7.42 P**
Silver nitrate, 4 grains.....	4.08	**3.61 P**	0.70	**0.90**	Not made	
Sodium chloride, 0.9% solution...	6.42	**6.75 M**	6.50	**6.88 P**	7.32	**7.45 M**
Zinc chloride, 1 grain.....	5.70	**5.15 M**	6.50	**4.80**	6.08	**6.60 P**

* In this table and table 2 M indicates mold and P, precipitate.

in all cases except one, that of the sodium biborate eye wash (made of sodium borate, camphor and water). It is interesting to note that in the case of each of the cork-stoppered bottles either mold or a precipitate developed in the medicine, as against only three bottles with screw caps. This formation of precipitates and mold was also noted to a great extent in the preparations made with the buffer solution. It was because of this undesirable sediment that I soon made the change to bottles with droppers, for dispensing ophthalmic medicaments. The change in p_H after two months of opening the bottles, as shown in table 1, was least in preparations made with the buffer solution, even though cork stoppers were used. It would seem, therefore, that the best way to dispense the medicaments is in buffer solutions and in bottles with screw caps or droppers.

BUFFER SOLUTIONS IN OPHTHALMOLOGIC PRACTICE

Buffer solutions are useful in ophthalmologic practice because they insure uniformity of product and are quite stable. They are, in the great majority of cases, more easily tolerated by the eye than are nonbuffered solutions. The buffered solution is a vehicle the p_H of which can be altered to suit the needs of the patient, the active drug contained always remaining the same. For example, when bacteria can thrive only in an acid medium, the buffer is made alkaline, or the reverse.

Since the publication on buffers by Gifford and Smith,[11] I have used the buffer solutions which they recommended, with satisfactory results. Their combination buffer solution is a modification of that suggested by Atkins and Pantin[12] in 1926, being a buffer solution used for the testing of the p_H of sea water. For about a year, however, I have used exclusively a buffer solution recommended by Palitzsch.[13] It is simply made and is well tolerated by the eye. Its virtue lies in the fact that it contains an acid and the particular salt of that acid. It contains three well known bland and familiar medicaments, each of which has been used singly or in combination in ophthalmology, viz., boric acid, sodium borate and sodium chloride. The boric acid acts at the same time as a preservative and a mild antiseptic. Two parent solutions are made. One is a twentieth-molar solution of sodium borate, containing 19.108 Gm. of sodium borate to the liter. The other is a fifth-molar solution of boric acid, containing 12.404 Gm. of boric acid with 2.925 Gm. of sodium chloride. By the admixture of various proportions of the two parent solutions a number of different buffer solutions suitable for vehicles are obtained. By adjusting different proportions of the two parent solutions Palitzsch made seventeen different vehicles with a p_H ranging from 6.77 to 8.69. Each vehicle has a therapeutic value because of the ingredients (boric acid, sodium borate and sodium chloride). The aforementioned combinations of buffer solutions can be used in dispensing all preparations except in the cases of butyn, zinc and cocaine or other substances which require definite acid vehicles. In prescribing these the mixture of a fifth-molar solution of boric acid with sodium chloride can be employed.

The most common buffer vehicles which I have made and used are as follows:

A buffer solution of p_H 6.77, made by combining 0.3 cc. of a solution of sodium borate with 9.7 cc. of a solution of boric acid and sodium chloride.

12. Atkins, W. R. G., and Pantin, C. F. A.: A Buffer Mixture for the Alkaline Range of Hydrogen Ion Concentration, Biochem. J. **20**:102, 1926.

13. Palitzsch, S.: Manuel pratique de l'analyse de l'eau de mer, Bull. Inst. océanog., Monaco, no. 409, April 1922.

A buffer solution of p_H 7.2, made by combining 1 cc. of a solution of sodium borate with 8.5 cc. of a solution of boric acid and sodium chloride.

A buffer solution of p_H 8.69, made by combining 6.0 cc. of a solution of sodium borate with 4 cc. of a solution of boric acid and sodium chloride.

A 3 per cent solution of glycerin, as recommended by Smith,[14] the boric acid-sodium borate buffer of p_H 7.2 being used.

I have taken the last-named four vehicles and have added to them different medicinal agents. In each case only one ophthalmic drug was mixed with the vehicle of known p_H. After the addition of the drug

TABLE 2.—*p_H of Solution After Drugs Were Added to the Buffer Vehicles*

	Boric Acid-Sodium Borate Buffer, p_H 6.77		Boric Acid-Sodium Borate Buffer, p_H 7.2		Boric Acid-Sodium Borate Buffer, p_H 8.69		3 per Cent Glycerin in Boric Acid-Sodium Borate Buffer, p_H 7.2; Final p_H, 7.00	
Drug Used, 1 Oz. Mixtures	First Reading	Reading After 2 Mo.	First Reading	Reading After 2 Mo.	First Reading	Reading After 2 Mo.	First Reading	Reading After 2 Mo.
Atropine sulfate, 4 grains	6.90	**6.60**	7.38	**7.00**	8.70	**8.00**	6.91	**6.69** M
Phenacaine hydrochloride, 1% solution	6.50	**6.50**	Not made		8.62	**7.95**	Not made	
Homatropine hydrobromide, 1 grain	6.90	**7.40**	7.31	**7.70**	8.65	**8.20**	6.90	**7.48** M
Pilocarpine, 1 grain	6.00	**6.05**	7.18	**7.10**	8.05	**7.68**	6.60	**6.90** M
Physostigmine, 1 grain	6.80	**6.70**	7.30	**7.20**	8.65	**8.42**	6.83	**6.80** M
Physostigmine, 2 grains	6.74	**6.60**	7.25	**7.01**	8.62	**8.22**	Not made	
Metaphen, 1:5,000 dilution	7.00	**6.56**	7.42	**7.13** P	8.70	**8.90** P	7.20*	
Metaphen, 1:10,000 dilution	7.00	**6.23**	7.30	**6.80**	8.71	**8.63**	7.05*	
Zinc sulfate, 1 grain	7.09	**6.78** P	7.09	**6.78** P	8.32	**8.11** P	6.85*	

* Congealed at 40 F. (5 C.).

the p_H was again determined. The results are given in table 2. The bottles were opened for ten minutes daily for a period of two months, as in the tests the results of which are shown in table 1. After this period the p_H was again determined. It is noted in boldface type to the right of the original p_H. The bottles used in all these tests had droppers. It will be noted from table 2 that on the addition of a 3 per cent solution of glycerin to the boric acid-sodium borate vehicle the p_H dropped from 7.2 to 7. It is seen that in the great majority of cases the solution resulting from the addition of the drug to the buffer vehicle tended to show a slight rise in p_H. The opposite tendency is noted to a slight extent in the vehicle of p_H 8.69 and in the vehicle of p_H 7 with a 3 per cent buffer solution of glycerin.

14. Gifford, S. R.: A Handbook of Ocular Therapeutics, Philadelphia, Lea & Febiger, 1932, p. 55.

After two months of daily opening of the bottles for ten minutes at a time to simulate the amount of daily exposure to air that might occur in the ordinary use of a medicament, only metaphen in a 1 : 5,000 dilution at p_H 7.13 and 8.90 showed a precipitate. As zinc and phenacaine hydrochloride require an acid medium, precipitations with these were not unexpected. Molds were found in all the mixtures of glycerin. The mixture of metaphen and glycerin congealed when the temperature of the room dropped to 40 F. (5 C.). This occurred also in the case of the mixture of zinc and glycerin.

Ordinarily, eucatropine (a 5 per cent solution) is said to require an acid vehicle for making a pure solution. I have been able to use a boric acid-sodium borate buffer of p_H 7 with this drug and yet have complete solubility. No precipitate formed on standing.

Tice,[15] in a review of an article on the preparation of eye drops by Buchi and Baeschlin, reported their successful use of boric acid-sodium borate buffer solutions and their methods of also utilizing the parent mixture of boric acid to include those drugs not soluble in alkaline or neutral solutions.

The buffers of a p_H ranging from 7 to 8 have proved effective for solutions which I used with contact lenses. The burning sensation experienced with contact lenses after wearing them for a short period when using physiologic solution of sodium chloride (p_H 6.4 to 6.5) or Ringer's solution (p_H 7.45) is not noted so soon when a buffer solution of p_H 7 to 8 is used. Ringer's solution gives a better result than does physiologic solution of sodium chloride, but is not as good as the buffer solution. This was verified in a number of instances. In one outstanding case a patient who could not retain a contact lens for more than one hour when physiologic solution of sodium chloride was used or for over two hours when Ringer's solution was used was able to use the contact lens comfortably for ten hours when a buffer solution was used. I believe this increased tolerance was made possible, first, by the medicaments comprising the buffer solution and, second, by the fact that this solution can be so altered as to make its p_H one which is pleasant to endure.

That the p_H of the fluid in a contact lens does not change materially has been borne out by the experiments carried out in the following cases.

CASE 1.—M. S. had a conical cornea in each eye. Tears were examined. Those of the right eye had a p_H of 7.25, and those of the left eye, a p_H of 8.05. A contact lens was inserted on each eye, a buffer solution of p_H 7.3 being used. The eyes became irritable after ten hours. When the contact lens was removed, the fluid in the contact lenses was examined. The solution in the lens for the right eye showed a p_H of 7.33, and that in the lens for the left eye showed a p_H of 7.03.

15. Tice, L. F.: The Preparation of Eye Drops, Am. J. Pharm. **108**:308, 1936.

CASE 2.—A. K. had high myopia. Tears could not easily be obtained. A contact lens was inserted in each eye, a buffer solution of p_H 7 being used. After two hours the eyes became irritable. The p_H of the buffer solution in each eye was 7.12. At another date a second test was carried out on the same patient. The p_H of the tears, which were then easily obtained, was 7.25 for the right eye and 7.05 for the left. A contact lens was placed on each eye, a buffer solution of p_H 7.53 being used. The lenses were worn about three hours and were removed on signs of oncoming irritation. The solution in the lens for the right eye showed a p_H of 7.52 and that in the lens for the left eye a p_H of 7.76.

The use of physiologic solution of sodium chloride with contact lenses in a few cases did not change the p_H more than 0.5. Of course, if the contact lens could have been worn longer, as is the case when a buffer solution is used, the p_H might have shown greater variations.

After the contact lens was worn for a while the eye became irritable in much the same manner as though there was a foreign body in the eye. Many patients, after wearing the contact lens for some time, in addition to irritation, noted a blurring in vision. With the thought that there might be a difference in the refractive index of the various solutions used, which, in addition, might cause ocular fatigue and irritation, Dr. Arthur Osol, of the Philadelphia College of Pharmacy and Science, assisted me in checking the refractive indexes of three solutions. The following almost identical results were obtained for all three mediums: The refractive index of the boric acid-sodium borate buffer of p_H 7.2 was 1.33416; that of the physiologic solution of sodium chloride of p_H 6.5 was 1.33405, and that of the triple-distilled water of p_H 7.1 was 1.33246.

I have used buffered solution of the boric acid-sodium borate type in a few hundred cases of extra-ocular disease. Only two of the patients reported that the results were not satisfactory. I believe that if these buffers were adjusted to the particular patient this shortcoming would be remedied. Ordinarily a p_H of from 7 to 7.3 was used. The startling improvements which I have obtained in acute and chronic external ocular infections have amply repaid me for the trouble and painstaking care required in the making of the buffer solutions.

SUMMARY AND CONCLUSIONS

The value of p_H in industry and science is briefly considered. A graphic description of calibrating p_H as devised by Sorenson is given.

The chief methods of determining p_H are described briefly, with particular reference to the electrometric or potentiometric test. The means of studying the p_H of small quantities of solution (e. g., tears) by the use of the Cullen tube is given.

It was shown that distilled waters vary in p_H. Therefore the same medicaments made with different distilled waters were found to vary.

The p_H of the tears of the same person in either health or disease may be different from time to time. Nor is the p_H of the tears of the two eyes always the same. The average range of the p_H of tears is between 7 and 7.5. Persons with extra-ocular diseases do not show typical changes in the p_H of the tears.

Buffer solutions are defined, and various examples of those which may be used in ophthalmology are given. The advantages of dispensing ophthalmologic prescriptions in bottles with a screw cap or dropper tip as against cork-stoppered bottles are shown.

Preparations made with a buffer solution as a vehicle are more stable and more soothing to the eye than those made with distilled water, owing to the medicinal properties of the ingredients of the buffer solution. The buffer solutions are mildly antiseptic and contain preservatives. The change in p_H of buffer solutions caused by the addition of various drugs used in diseases of the eye has been shown after compounding the preparation as well as after the simulated use of such preparations for some time.

The better results obtained by using buffer solutions instead of physiologic solution of sodium chloride or Ringer's solution for contact lenses is noted.

Buffer solutions can be used in counteracting extra-ocular infection by creating a reaction of the tears which is inhibitive to the growth of bacteria.

Miss Evelyn M. Carpenter and Service B of the ophthalmologic department of the Protesant Episcopal Hospital gave the financial aid necessary to make this study, and B. H. Hoffstein, formerly associated with the Philadelphia College of Pharmacy and Science, assisted in solving some of the pharmaceutical problems.

LESIONS OF THE FUNDUS IN POLYCYTHEMIA

REPORT OF CASES

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Polycythaemia vera is generally regarded as a clinical entity caused by primary hyperplasia of the erythroblastic elements of the bone marrow. Its origin is unknown. It is seen infrequently by the internist and even more rarely by the ophthalmologist, as vision is seldom disturbed. Examination of the fundus, however, is likely to reveal early characteristic lesions which are of value in the diagnosis. In view of these considerations I have decided to present this subject, together with a brief report of a few cases.

Although the term polycythemia signifies an increase in the cellular elements of the blood, it is now generally applied to an increase in the number of red blood cells or erythrocytes; therefore the disease is often called erythremia. Vaquez [1] in 1892 was the first to describe polycythemia, in an article entitled "Cyanosis with Polycythemia," while William Osler [2] in 1903 presented the well known and important paper entitled "Chronic Cyanosis with Polycythemia and Enlargement of Spleen." Hence the disease is also known as the Vaquez-Osler disease. The lesion of the fundus was called by Ascher fundus polycythemicus. As early as 1859 Hermann Knapp [3] presented before the Naturhistorisch-medicinischer Verein of Heidelberg a report of a case entitled "A Case of Hyperemia of the Retina with Choked Disk." U. Kümmel [4] stated that he considered this case one of polycythemia. More recently articles on this condition have been published by Julius Ascher,[5] Edward Jackson,[6] Carl Behr,[7] G. E. de Schweinitz and Alan Woods [8] and others.

Read at the regular meeting of the Section of Ophthalmology of the New York Academy of Medicine, Dec. 21, 1936.

1. Vaquez, H.: Cyanosis with Polycythemia, Bull. méd. **6**:849, 1892.
2. Osler, William: Chronic Cyanosis with Polycythemia and Enlargement of Spleen, Am. J. M. Sc. **126**:187, 1903.
3. Knapp, H.: A Case of Hyperemia of the Retina with Choked Disk, Verhandl. d. naturh.-med. Ver. zu Heidelberg **3**:84, 1859-1862.
4. Kümmel, U., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1931, vol. 7, p. 73.
5. Ascher, J.: Klin. Monatsbl. f. Augenh. **53**:388, 1914.
6. Jackson, E.: Tr. Am. Ophth. Soc. **11**:363, 1906.
7. Behr, C.: Klin. Monatsbl. f. Augenh. **49**:672, 1911.
8. de Schweinitz, G. E., and Woods, A.: Tr. Am. Ophth. Soc. **23**:90, 1925.

Recent studies in hematology with reference to polycythemia will probably change some of the earlier diagnoses with respect to cyanosis retinae, a term which, according to Posey,[9] is applied solely in connection with heart disease. The case reported by Baquis [10] and that of H. H. Tyson,[11] both of which were published in 1908, might now be considered cases of fundus polycythemicus occurring in conjunction with congenital heart disease with associated secondary or compensatory polycythemia rather than cases of cyanosis retinae. The changes in the blood in both cases were typical of polycythemia; that is, the red blood cell count was over 8,000,000 and the hemoglobin content was over 100 per cent in each case. Both patients suffered from congenital heart disease associated with splenic enlargement. Carl Behr [7] regarded the condition in these cases as polycythemia. Cyanosis retinae is due to general cyanosis found in cases of polycythemia, heart disease and many other conditions; for this reason I believe that it should not be used solely in reference to heart disease.

There are two types of polycythemia. The primary form, or polycythaemia vera, the cause of which is unknown, is characterized by an increase in the number of red cells, the hemoglobin content and the volume of the blood and is accompanied by headaches, malaise, neurologic symptoms, general cyanosis and enlargement of the spleen. It generally occurs between the ages of 35 and 65. The secondary or compensatory form is usually due to congenital heart disease, although other causes are emphysema, stenosis of the pulmonary artery, and dehydration of the tissues (as in cholera and dysentery). A temporary form is found in persons living in high altitudes. It may occur at any age. The general manifestations of the secondary form of polycythemia are similar to those observed in the primary type, except that in the secondary type the cyanosis is apt to be more marked; the tongue and lips are more cyanotic; the finger tips are more clubbed, and the spleen is not usually enlarged. Either type may be associated with mild or severe ocular manifestations. At times it is possible to distinguish ophthalmoscopically between the primary and the secondary type, as reported by Engelking.[13]

Attempts to differentiate the ocular symptoms in polycythemia from those in cyanosis retinae have been reported in the literature. General cyanosis is due to deficient oxidation of the blood. This manifestation may be either mild or severe and may be associated with ocular lesions. If it affects the retina it is called cyanosis retinae and is confined chiefly

9. Posey, W. C.: Tr. Am. Ophth. Soc. **10**:634, 1905.
10. Baquis, E.: Arch. f. Ophth. **68**:177, 1908.
11. Tyson, H. H.: Arch. Ophth. **37**:555, 1908.
12. Footnote deleted.
13. Engelking, E.: Klin. Monatsbl. f. Augenh. **64**:645, 1920.

to the blood vessels, the surrounding tissue often not showing changes typical of cyanosis, owing to the light reflex from the retinal pigment. This cyanosis is sometimes most evident in the physiologic cup of the disk. In albinos with polycythemia a cyanotic appearance of the fundus could probably be more easily recognized on account of the lack of normal pigment. For severe cases in which other parts of the eye are involved Baquis has used the term cyanosis oculi. In some cases of severe congenital heart disease there is probably a compensatory reaction due to some interference with oxygenation, which stimulates the bone marrow and causes increased cyanosis with secondary polycythemia. I suggest that cyanosis retinae or cyanosis oculi should be considered in conjunction with other diseases rather than as an entity.

Another type of polycythemia is known as polycythaemia hypertonica, or Gaisböck's disease. This type is associated with arteriosclerosis, cardiac disease and renal disease and is accompanied by high blood pressure without splenomegaly.

The characteristic blood picture in polycythemia consists of an average high increase in the number of red blood cells from about 7,000,000 to 10,000,000, in conjunction with an increase in the hemoglobin content from about 110 to 126 per cent or over. The color index is low (less than 1), and the volume and viscosity of the blood are increased.

The fundus sometimes appears normal, while at other times lesions exist, which may be mild or severe, depending on the severity of the disease and whether or not the patient has responded to treatment. The existence of a high red cell count of the blood, with a corresponding increase in the hemoglobin content, frequently determines the degree to which the fundus is affected and also the extent of the vascular changes in the conjunctiva and iris.

The vascular lesion of the fundus is a part of the general vascular disturbance, which is not of an inflammatory nature, as proved by Carl Behr [7] in a report of ocular microscopic examination. The characteristic finding in the fundus in polycythemia is marked distention and engorgement of the retinal veins, which appear purplish. This distention is due mainly to an increase in the blood volume and a thinness of the venous wall. The change in color is caused by excessive replacement of oxygen by carbon dioxide. The resultant venous stasis is the basic factor in the causation of the ocular lesion.

Of the seven cases of polycythemia reported in this paper, five are cases of the primary type, and two are cases of the secondary type. In the latter type there were no fundic complications; the retinal veins were distended, slightly tortuous and bluish; the retinal arteries were moderately dilated and were purplish. In one case the disk appeared reddish and the fundus had a grayish tinge; in the other there was a

bluish appearance of the physiologic cup and the veins, while the color of the fundus seemed normal. In both cases the ocular and tarsal conjunctivae were congested and their veins dilated. Slit lamp examination showed that in one case the iris was normal and that in the other there were dilated iritic blood vessels with chocolate-colored areas and dustlike pigment scattered over some of the vessels. Vision and ocular function were normal in both cases.

Of the cases of the primary type, in two there were lesions of the fundus, while in three the lesions were solely vascular, consisting of distention of the retinal veins, which were purplish and moderately tortuous. In these three cases the arteries appeared normal in caliber and color, and the disk was also normal. The fundic complications in the two cases of primary polycythemia were as follows: One patient presented venous engorgement in one eye, with edema of the disk and retinal hemorrhages; the other showed bilateral postneuritic atrophy of the optic nerve, with distended veins which were bordered by a distinct broad whitish band along the perivascular space, due to a transudation of plasma. The perivascular space of the retinal arteries was not involved.

At the meeting the Section of Ophthalmology of the New York Academy of Medicine in 1918 I [14] presented a case of polycythemia complicated by unilateral thrombosis of the central retinal vein.

In the cases reported in this paper, vision was normal with each type of polycythemia, except in one case of the primary type. Visual disturbances were reported in 1919 by Friedenwald,[15] who expressed the opinion that the deterioration of vision noted in those of his patients who were without lesions of the fundus was caused by retrobulbar neuritis and papillitis.

It should be remembered that variations and congenital anomalies, as well as local lesions, may be present in the fundus. The general symptoms of the disease will aid in the diagnosis of the condition of the fundus.

Carl Behr [7] in 1911 published a report giving the pathologic findings in polycythemia, with illustrations of [illegible] dilated and engorged retinal and uveal vessels filled with numerous red and a few white blood cells. He noted also edema of the disk but no inflammatory reaction.

The prognosis of polycythemia depends on whether one is dealing with the primary or the secondary type. In cases of the former type the prognosis depends on the severity of the disease, some patients living to an advanced age. Death is caused by complications such as

14. Cohen, M.: Arch. Ophth. **47**:2, 1918.

15. Friedenwald, H.: Visual Disturbances in Polycythemia Vera, in Contributions to Medical and Biologic Research, dedicated to William Osler, New York, Paul B. Hoeber, Inc., 1919, vol. 1, p. 495.

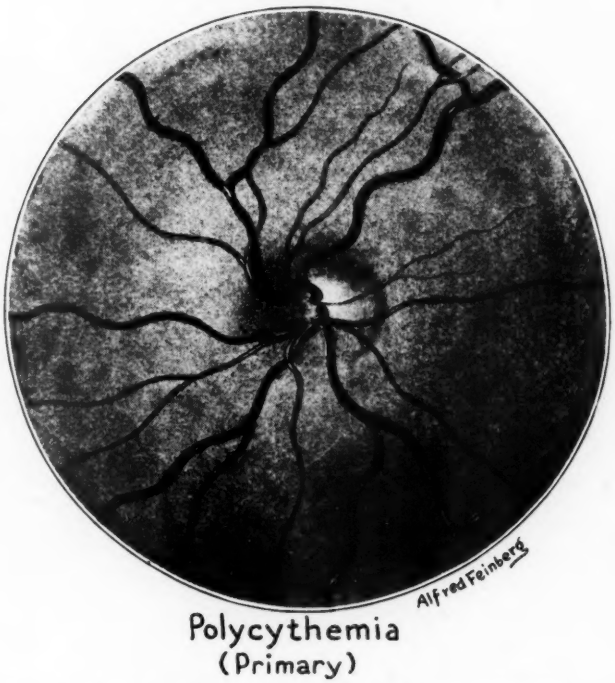

Fig. 1.—Lesion of the fundus in primary polycythemia.

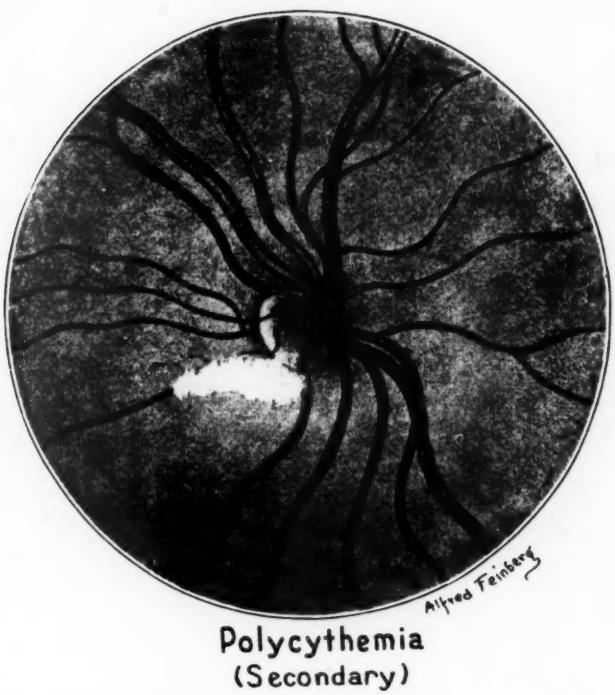

Fig. 2.—Lesion of the fundus in secondary polycythemia.

heart failure, hemiplegia, thrombosis and hemorrhages. In cases of the secondary type the prognosis depends on the severity of the cardiac lesion or other complicating disease.

Treatment differs for the two types. In cases of the primary type several methods of treatment have proved of definite value, including venesection, the use of phenylhydrazine and irradiation. In cases of the secondary type the causative disease is treated along the lines indicated by the condition.

Since few cases of polycythemia have been observed at the medical clinic and service of the New York Post-Graduate Medical School and Hospital, my associates and I were obliged to obtain additional patients for presentation. A brief report of these cases, including the average findings of the blood, follows.

REPORT OF CASES

CASE 1.—H. S., a man aged 64 years old, a factory worker, was admitted to the medical clinic of the New York Post-Graduate Medical School and Hospital on Feb. 27, 1932, complaining of vertigo. The history revealed no abnormality except neuralgic pains. The skin had been cyanotic for seven years. The patient seemed well nourished and did not appear to be acutely ill. The mucous membrane of the mouth and pharynx was bluish. The heart was normal in size; its sounds were of good quality, and there were no murmurs. The lungs were clear. The spleen and liver were moderately enlarged. The blood pressure was 126 systolic and 64 diastolic. The urine was normal. The red cell count of the blood was 9,040,000, with 130 per cent hemoglobin; the white cell count was 7,900. Roentgenograms of the long bones showed atrophic changes compatible with dyscrasia of the blood. Microscopic examination of the capillaries of the nail beds of the fingers showed them to be markedly dilated and packed with red cells, especially in the venous segment. The visual disturbances present were due to an incipient cataract in each eye. The findings for the fundus were: retinal venous distention with engorgement, the veins appearing purplish, and irregularity of the lumen of the superior temporal vein. The arteries and the disk were normal. Acetylphenylhydrazine was used in treatment, with remarkable results. The general diagnosis was primary polycythemia. The diagnosis of the condition of the fundus was fundus polycythemicus.

CASE 2.—A. B., a married woman 65 years old, was first examined at the New York Post-Graduate Medical School and Hospital in 1929, at which time she complained of vertigo. The following were noted: cyanosis of the hands and lips, of twelve years' duration; clubbing of the fingers, and pain over the heart, which was enlarged (a secondary condition). The urine showed occasional red blood cells. The red cell count of the blood was 8,152,000 with 110 per cent hemoglobin. The blood pressure was 196 systolic and 112 diastolic. The spleen and liver were enlarged. Vision was normal in both eyes with correction, and the field of vision was normal. Examination of the fundus showed distended and purplish veins; the arteries and disk were normal. The general diagnosis was primary polycythemia. The diagnosis of the condition of the fundus was fundus polycythemicus.

CASE 3.—O. C. M., a salesman 48 years old, was admitted to the New York Post-Graduate Medical School and Hospital on June 28, 1932, complaining of precordial pain (not radiating to the arms) and insomnia. The past history was negative. The face was deep red, and the mucous membranes had been purple for four years. The lungs were clear, and the heart was normal. The spleen was palpable. The blood pressure was 108 systolic and 82 diastolic. The red cell count of the blood was 8,000,000, with 154 per cent hemoglobin. The color index was 0.96. The oxygen capacity, oxyhemoglobin content, viscosity and blood volume were determined and considered as positive factors for the diagnosis of polycythemia. Vision in the right eye was 20/200 (the poor vision was due to congenital amblyopia); vision in the left eye was corrected to 20/20 with glasses. Examination of the fundus showed distended and purplish retinal veins, normal retinal arteries and a reddish disk. The general diagnosis was primary polycythemia. The diagnosis of the condition of the fundus was fundus polycythemicus.

CASE 4.—F. M., a man 34 years old, was admitted to the Manhattan Eye, Ear and Throat Hospital (in the service of Dr. David Webster) in January 1934. There was a history of poor vision and cyanosis for three years. The chief complaints at the time of admission were poor vision, and varices in both legs. Physical examination showed marked cyanosis of the face. The liver and spleen enlarged. Study of the blood (conducted at the New York Hospital) showed a red cell count of 5,500,000, with 110 per cent hemoglobin; the carbon dioxide content of the venous and arterial system was definitely increased; the oxygen capacity and the viscosity were normal. On admission, vision of the left eye was equal to perception of light, and vision of the right eye was 6/200. There was slight exophthalmos of both eyes, together with divergence of the left eye. Examination of the fundus showed bilateral postneuritic atrophy of the optic nerve, with distended purplish veins bordered by a distinct broad whitish band along the perivascular space, due to a transudation of plasma; the perivascular space of the retinal arteries was not involved. The general diagnosis was primary polycythemia. The diagnosis of the condition of the fundus was fundus polycythemicus associated with bilateral postneuritic atrophy of the optic nerve.

CASE 5.—A. B., a laborer 38 years old, was admitted to the medical ward of the New York Post-Graduate Medical School and Hospital on Jan. 10, 1935, complaining of severe headaches for one year and profuse diaphoresis. The past history was essentially unimportant. Examination showed definite cyanosis of the conjunctiva, lips and fingers. Examination of the capillaries of the nail beds of the fingers showed from two to three times the normal number in action, which is characteristic of polycythemia. Vision was normal in both eyes. The blood pressure was 130 systolic and 98 diastolic. The lungs were clear; the heart was normal; the spleen was not palpable. The red cell count of the blood was 7,800,000 with 142 per cent hemoglobin. The determination for the blood volume (per kilogram of body weight) totaled 104 cc. (the normal is from 82 to 88 cc.). The oxygen capacity of the blood was 29.3 volumes per cent (the normal is from 17.5 to 21.4 volumes per cent); the carbon dioxide content was 61.4 volumes per cent (the normal is from 48 to 64 volumes), and the oxygen content was 18.1 volumes per cent (the normal is 14.6 volumes per cent). Examination of the fundus showed distended purplish veins, edema of the disk and two small retinal hemorrhages in one eye. The general diagnosis was primary polycythemia. The diagnosis of the condition of the fundus was fundus polycythemicus, with edema of the disk.

CASE 6.—M. K., a boy 8½ years old, was a patient in the clinic of the Gouverneur Hospital. Symptoms of heart disease had been present for five

years. There were marked cyanosis of the lips and tongue and clubbing of the finger-tips. The patient complained of dyspnea and fatigue on exertion. The spleen and liver were not palpable. The red cell count of the blood was 9,300,000, with 160 per cent hemoglobin; the white cell count was 10,000. The blood pressure was 80 systolic and 50 diastolic. Vision was normal in both eyes. Examination of the fundus showed distended, slightly tortuous, bluish retinal veins; moderately dilated, purplish retinal arteries; a reddish disk, and a fundus with a grayish tinge. The general diagnosis was congenital heart disease (pulmonary stenosis) associated with secondary or compensatory polycythemia. The diagnosis of the condition of the fundus was fundus polycythemicus.

CASE 7.—F. D., a married man, 32 years old, was admitted to the clinic of the New York Post-Graduate Medical School and Hospital in 1929, complaining of vertigo. He had blueness of the face, lips and tongue. There had been shortness of breath for ten years, and clubbed fingers were noted. The blood pressure was 102 systolic and 76 diastolic. The spleen was not palpable. The red cell count of the blood was 9,790, 000, with 130 per cent hemoglobin. The color index was 0.91. Vision and the field of vision were normal. Examination of the fundus showed distended, slightly tortuous, bluish retinal veins and moderately dilated, purplish retinal arteries. The physiologic cup was bluish; the color of the fundus was normal. The general diagnosis was congenital heart disease associated with secondary or compensatory polycythemia. The diagnosis of the condition of the fundus was fundus polycythemicus.

CONCLUSIONS

Cyanosis retinae is associated with many diseases. For this reason it is suggested that the term should not be limited exclusively to the condition associated with congenital heart disease but should be applied to the condition in any disease, including polycythemia, as this disease is accompanied by cyanosis.

Seven cases of polycythemia are reported.

In the five cases of polycythemia of the primary type the retinal veins were distended and purplish, and the retinal arteries were of normal caliber and color. Complications occurred in two cases. Vision was normal in all cases except one.

In the two cases of polycythemia of the secondary type the retinal veins were markedly distended and were bluish, while the retinal arteries were moderately distended and were purplish. There were no fundic complications, and vision was normal in both cases.

The differences in the fundus in the two types of polycythemia corresponded with the findings already reported in the literature.

Ophthalmoscopic examination in the early stages of the disease is an additional aid in the diagnosis of both types of polycythemia.

It is essential that a general physical examination be made in order to establish the diagnosis of fundus polycythemicus, as at times the vascular lesion of the fundus is not apparent.

Drs. Dan Witt, H. O. Mosenthal and Moses Deren made the general medical examination in the cases reported.

SHORT STUDIES ON THE HISTORY OF OPHTHALMOLOGY

IV. SIR CLIFFORD ALLBUTT, THE APOSTLE OF MEDICAL OPHTHALMOSCOPY

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PHILADELPHIA

On Feb. 22, 1925, there died in England that "spiritual aristocrat of Medicine," Sir Thomas Clifford Allbutt, Regius Professor of Physic of the University of Cambridge. Sir Clifford, although famous throughout the last fifty years of his life for his teaching in general medicine and long the leader of the medical profession in England, was also, it should be remembered, one of the most valuable contributors to ophthalmologic knowledge, one of the first to use the ophthalmoscope and to extend its use among general practitioners.

Clifford Allbutt, the only son of the Reverend Thomas Allbutt, was born at Dewsbury, near the city of York, on July 20, 1836. He had only a younger sister. When a small boy he became the child companion of an invalid lady, a cousin of his father, on an extended visit to her at Ventnor on the Isle of Wight. There he had some private instruction. His formal education began later in St. Peter's School in York, founded in the seventh century, at which the famous Alcuin was once master. He left St. Peter's school in 1855, and, having gained a classical scholarship, was admitted to Gonville and Caius College of the University of Cambridge, from which he was graduated with high honors in the first class of natural science established there.

"Gonville and Caius" is the college at which scientific and medical courses are held. This illustrious college was incorporated in 1557 by Dr. John Keys, whose name was latinized as Caius, one of the great physicians of the sixteenth century; he served King Edward VI, Queen Mary and Queen Elizabeth and was president of the Royal College of Physicians. Caius had studied at Padua, becoming a physician in the faculty in 1541, and for two years lived with Vesalius. He returned to England at the request of Henry VIII and lectured on anatomy to the surgeons of London for twenty years thereafter. He was not only a great naturalist, the first English anatomist, but a great physician, besides being a distinguished antiquary and classical scholar. He, like Linacre, of Oxford, was a disciple of the new learning brought from Italy. "Caius" has always been a physicians' college; Harvey, Glisson,

Fig. 1.—Two views of Gonville and Caius College, Cambridge University showing the Gate of Virtue.

the anatomist, and a long roll of eminent surgeons and physicians received their education there. Caius was master of the college of which he was the cofounder and generously contributed to its maintenance. The foundation stone he had himself inscribed in Latin, the equivalent being "John Caius dedicates this to Knowledge." He is buried in the chapel.

Dr. Caius, more than three hundred years in advance of the time, appreciated the importance of sun and air to the living organism by decreeing in the statutes of Caius "that no building shall be constructed which shall shut off the entire south side of the College lest for lack of

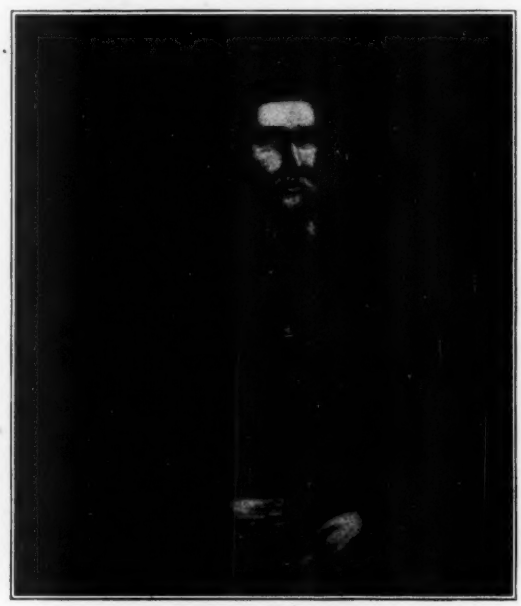


Fig. 2.—Portrait of Joanes Caius.

free ventilation the air shall become foul, and the health of the College impaired and disease and death ensue." Closed quadrangles had previously been built at all the colleges, but no more were built after the time of Caius. Allbutt must have been deeply impressed by the facts of the life of the founder of his college and must have been actuated to an emulation of Caius' activities.

When Allbutt entered Cambridge University his tastes and inclinations were more literary and artistic than scientific. Besides being a serious student of music, he became a pre-Raphaelite; later, when he visited Italy, he aspired to be an artist, but as he lacked the power of

expressing himself in painting he abandoned that desire. While at Caius his reading naturally widened, and he was impressed by the philosophy of Auguste Comte, so much that he decided on medicine as a career.

In his boyhood he was attracted to things medical by his visits to two uncles of his in the neighborhood who were physicians; they allowed the lad free access to their old-fashioned surgeries, where he learned the names and uses of the various drugs on their shelves. He had formed a museum of his own at one time, but all those interests were forgotten during his classical studies; they were revived, however, as has already been stated, by the contemplation of the discoveries which were then being made in biology, and through such influences he was led to begin the study of medicine seriously. Accordingly, he was entered at St. George's Hospital, London, where he had the good fortune to receive instruction from such illustrious teachers as Bence Jones, the pupil of Liebig, Sir George Paget and Sir Humphrey Murray. The inspiration exerted by these teachers endured throughout his lifetime and led him on to further study. By having taken the full curriculum and much extra research he was enabled to receive at Cambridge the degree of Bachelor of Medicine in 1861; but he did not receive his degree of Doctor of Medicine till 1869.

Following the advice of Bence Jones, whose clinical clerk he became, after receiving his baccalaureate Allbutt went to Paris, where, allying himself with Trousseau, of the faculty of the University of Paris, he began the study of the diseases of the heart and blood vessels, and, in association with Duchenne, began the observation of nervous diseases. Fascinated by certain aspects of dermatology, he attended the course given with great charm by Bazin. He returned to England by the end of the year and resumed his studies, especially under the direction of Sir William Jenner. Already he had shown evidences of the excellent tutoring that he had received and began to manifest an interest in pathology, especially the pathology of the nervous system, and through the guidance of Dr. John W. Ogle, the same teacher who had so greatly influenced Hughlings Jackson, he became seriously interested in a course in ophthalmology.

In 1861 Allbutt entered into practice in Leeds, in which city of woolen mills he was to reside for twenty-eight years. He attached himself to several institutions, among which were the House of Recovery, one of the early "fever-hospitals" in England, and the Leeds General Infirmary, to which he was appointed physician. His success was immediate. Two years later he received the appointment of lecturer on anatomy, comparative as well as human. He continued his association with the Leeds General Infirmary for twenty years and became one of a band of physicians remarkably distinguished in medi-

cine and surgery. Leeds was noted more for surgery than for internal medicine. Allbutt became allied especially with Mr. T. Pridgin Teale Jr., who later became a well known ophthalmic surgeon.

OPHTHALMOLOGIC INTERESTS

As has been stated already, Allbutt, while at St. George's Hospital, by his association with Dr. John W. Ogle, became interested in pathology, especially the pathology of the nervous system. During his conferences Allbutt received from Ogle the idea of the value of the ophthalmoscope in disease of the central nervous system. It is well to be reminded that it was due to his acumen that Ogle, soon after it had

Fig. 3.—Leeds Infirmary, 1867.

been discovered, became convinced of the possible usefulness of the ophthalmoscope. In a paper which he published in 1860 Ogle[1] set forth the value of ophthalmoscopy in the study of disease of the brain and drew attention to "the very close relation which exists between the cerebral and the intraocular circulation." And he predicted that "the vascular structure of the posterior parts of the eye might serve in its variations as an index to the vascular conditions of the intracranial organs."

As Mr. Teale and Dr. Allbutt worked together at the infirmary on diseases of the eye, the former from the special side of the study and the latter from the more general aspect, they brought under observa-

1. Ogle, John W.: M. Times & Gaz. **1**:572, 1860.

tion a great number of important cases in which the patients presented the ocular disease coincidental with other diseases, which otherwise would have been overlooked. Allbutt was aware that changes might exist within the eye along with disease of other organs, but as yet the laws of their occurrence were not known.

Mr. Teale had become expert in the use of the ophthalmoscope and, naturally, constantly employed it in his ophthalmic clinic. He had had forms made on which the fields of vision could be readily mapped, which maps taken at various periods of the same case he and Allbutt found often presented curious departures from what was at that day considered to be the normal.

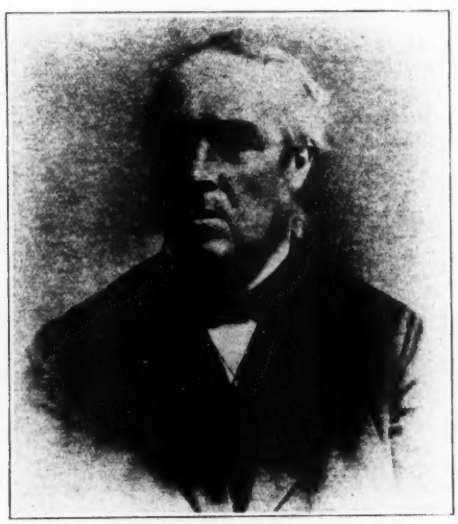


Fig. 4.—Dr. John W. Ogle.

These two, colleagues and confrères, began an association which lasted for many years, both living in friendly intercourse well beyond their octogenarian days. It is to that fellowship that ophthalmology in the north of England owes its foundation in scientific principles and its establishment as an integral part of the practice of medicine. They early began in reporting cases from their joint practice to describe the changes within the eye found accompanying disease of other parts of the body, particularly of the central nervous system. Their articles included papers on locomotor ataxia, epilepsy in association with disease of the optic nerve, atrophy of the optic nerve dependent on orbital disease, retinal diseases in association with disease of the kidney and atrophy of the optic nerve following typhus.

Mr. Teale gave to Allbutt "of himself, his experience and his materials so generously and so continuously, that Allbutt could no

longer define between that which was his own and that which really belonged to Mr. Teale." In various other ways these two physicians were associated throughout their long lives until the death of Teale separated them in 1924. Sir Clifford, in a letter to me shortly after that event, remarked that his "dear old colleague Pridgin Teale" had recently passed away, aged over 90, "bright up to the last."

Among the friends of Allbutt in Leeds was Dr. James Crichton-Browne. Only a short time before Allbutt's advent this physician had been appointed medical director of the West Riding Hospital for

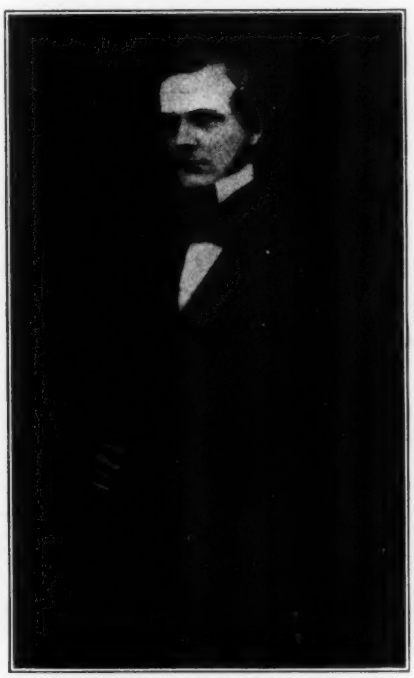


Fig. 5.—T. Pridgin Teale Jr., from a photograph taken about 1860, presented by his son, Mr. Michael A. Teale.

the Insane, located at Wakefield, about 10 miles (16.1 kilometers) from Leeds, containing about 1,500 beds. The enthusiastic director attracted there young men eager and keen like himself, and every facility for research was afforded them. In this band were David Ferrier, Hughlings Jackson, William Turner, Lauder Brunton, Milner Fothergill and others. Associated with them was Mr. Swanzy, then rising with distinction in ophthalmology. For years this band maintained their forces and pushed on together. Dr. Crichton-Browne, now Sir James, is still living, well beyond 97 years of age, enjoying an active intellectual life at Dumfries, on the east coast of Scotland.

It was not until several years after the invention of the ophthalmoscope that this instrument was much used. Mr. Spencer Wells,[2] in 1853, was among the first in England to insist on the great value of the ophthalmoscope in diseases of the eye. In their enthusiasm over the possibility of at least viewing the ocular fundus many who employed it were oversanguine as to the meaning of the appearances of the fundus in their relation to the brain. The ophthalmoscope was hailed as a sure means of making an accurate diagnosis as well as an advance in

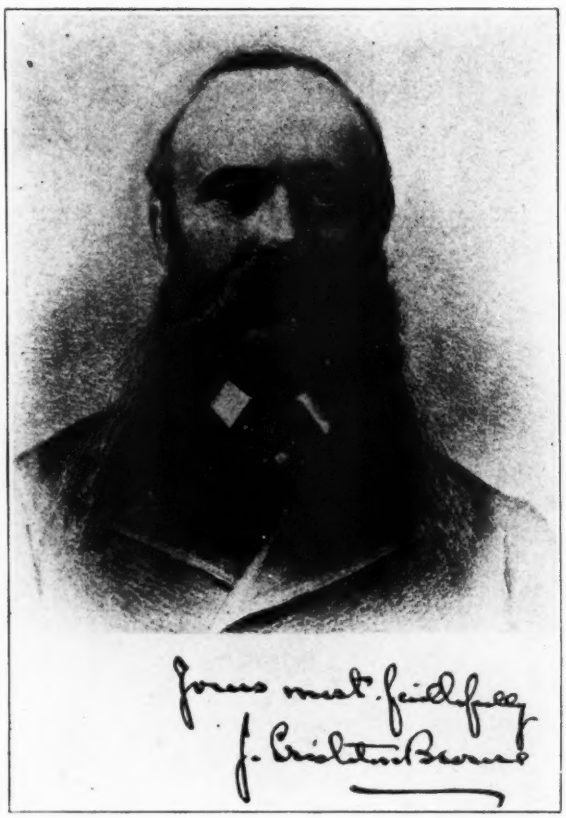


Fig. 6.—Dr. James Crichton-Browne in early years.

the understanding of the pathologic features of general diseases, thus serving as an effectual aid in therapeusis. Its advocates saw that the instrument would make possible an improvement over the methods that were then available in the diagnosis of cerebral troubles and that it would provide the means to discover the retinitis dependent on renal and other remote disorders. After a while reaction set in, and expectations began to be disappointed. Physicians unskilled in the use of the instru-

2. Wells, Spencer: M. Times & Gaz. **7**:264 (Sept. 19) 1853.

ment and unable to interpret the images revealed to them began to make blunders, so it was laid aside and, for a time, neglected. In the meantime a few patient workers toiled steadily in collecting data, comparing evidence and eliminating sources of error. Prominent among these workers was Clifford Allbutt, who, at the meetings of the various medical societies and in the columns of the *Lancet* and other journals, methodically explained the use of the ophthalmoscope, defined the range of its usefulness and described the phenomena made apparent by it.[3]

Fig. 7.—Spencer Wells.

After a year or so, however, Allbutt was gratified to learn that the ophthalmoscope was being more and more used by physicians. He was always careful to announce that it was Dr. John W. Ogle who had first drawn attention to the value of the instrument to physicians. The wide district from which the Leeds General Infirmary was supplied and the number of outpatients soon gave opportunities for such investiga-

3. Allbutt, Thomas Clifford, obituary, Lancet **1**:461 (Feb. 28) 1925.

tion. In spite of this practice Allbutt declared in his monograph published eighteen years later that "the number of physicians who are working with the ophthalmoscope in England may be counted upon the fingers of one hand. Many practitioners shrunk from the use of the ophthalmoscope because they believed that a troublesome arrangement of light and darkness was a necessary condition." It would seem from this backwardness that ophthalmoscopy was looked on as a difficult procedure not easily carried out.

As a matter of routine Allbutt examined the eyes of his patients in the hospital, and he hoped that his method might soon be used by physicians generally. He sought to simplify his examinations and to make

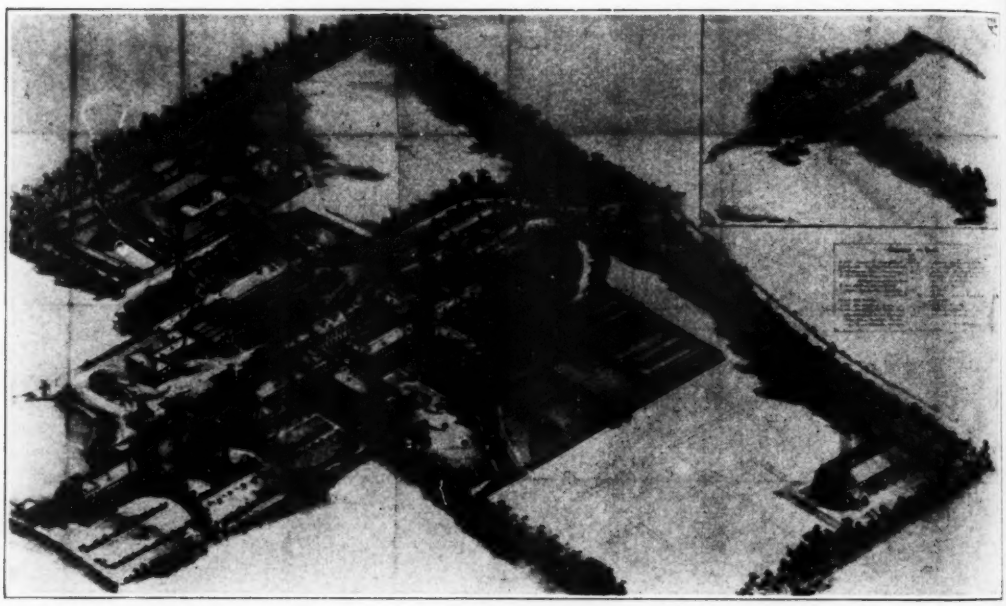


Fig. 8.—The West Riding Lunatic Asylum.

his explanations easy for beginners. He prepared diagrammatic outlines of the fundus on which could rapidly be sketched accurate representation on the state of any particular disk or retina. He endeavored to group together the patients with normal and healthy eyegrounds in order that his students might learn to define the natural details before passing on to other groups in which the picture was expressive of a departure from the normal and to groups with pathologic anomalies.

It was not long before Allbutt was able to arrange the details of his studies so systematically that they served as material for a course of lectures on "Optic Neuritis as a Symptom of Disease of the Brain and Spinal Cord." But as a preliminary to this formal presentation he recounted in much detail the analysis of his observations on a series of

several varieties of nervous diseases which he made in the examination of patients under Dr. Crichton-Browne's care. This was delivered under the title "The State of the Optic Nerves and Retina as Seen in the Insane" at a meeting of the Royal Medical and Chirurgical Society on Tuesday, Feb. 23, 1868.

While at Wakefield, Allbutt became greatly attracted to Hughlings Jackson, "whose simplicity, truthfulness and acuteness in the observation of disease and whose genuine insight into facts so long obscured by the cumbrous and vain phraseology of the Schools made Jackson's friendship as valuable to Allbutt as a student of nature, as the like qualities in his personal character won Allbutt's warm regard as his friend." Thus it was that Hughlings Jackson, in London, and Allbutt, in Leeds, began to use the ophthalmoscope in the investigation of cerebral disease at about the same time. That common interest procured for Allbutt the pleasure and the advantage of Jackson's friendship.

By 1867 Allbutt had accumulated from his experience sufficient material to publish a paper under the title "The Ophthalmoscope in the Physicians' Practice at the Leeds Infirmary."[4] This paper created much comment and was followed the next year by "Optic Nerves and Retinas of the Insane,"[5] "Medical Ophthalmology,"[6a] lectures on "Optic Neuritis,"[6b] "The Use of the Ophthalmoscope in Tubercular Meningitis and Spinal Disease"[7] and "Optic Neuritis in Pyaemia."[8]

In the preparation of these papers was laid the foundation for a remarkable monograph which appeared four years later, "On the Use of the Ophthalmoscope in Diseases of the Nervous System and of the Kidneys; Also in Certain Other General Disorders." While this work dealt with the subjects of the earlier special papers, nothing was transferred from them into the book, the first issued in English on the subject of ophthalmoscopy. In this work Allbutt expressed his own conclusions based on the vast amount of material which he had been gathering. It is interesting to note the list of the subjects presented.

After the "Aspect, Structure, and Connections of the Normal Optic Nerve and Retina," are dealt with "Variations from Health" are described. Then Allbutt defined "The Relations Between Certain Intracranial Disorders and Affections of the Optic Nerve and Retina," which was followed by a description of the "Ophthalmoscopic Signs of Diseases of the Spine," and retinitis dependent on albuminuria, on leukemia and on syphilis, each exhaustively described. Following these

4. Allbutt, Thomas Clifford: M. Times & Gaz. **1**:494, 1867.
5. Allbutt, Thomas Clifford: Med. Chir. Soc. London **51**:97, 1868.
6. Allbutt, Thomas Clifford: M. Times & Gaz. (*a*) **1**:495, 521, 574 and 628, 1868; (*b*) **2**:61 and 116, 1868.
7. Allbutt, Thomas Clifford: Lancet **1**:596, 1869; **1**:76, 1870.
8. Allbutt, Thomas Clifford: M. Times & Gaz. **1**:691, 1868.

are chapters on the amaurosis of diabetes and of oxaluria and on the toxic amauroses. An interesting chapter details the "Effects of Disorders of the Menstrual and Other Secretions upon the Nerve and Retina." The final chapter tells of "Embolism of the Central Artery of the Retina and Its Branches." In an appendix are recounted numerous histories describing the ophthalmoscopic changes which Allbutt had observed in cases of insanity in association with epilepsy, and of mania in association with idiocy and in association with "general paralysis" (dementia paralytica). In all these cases the author confined himself to those changes which he had looked on as having a symptomatic value, making no attempt to describe evidences of local disease.

In the preparation of his work on the ophthalmoscope, Allbutt was thoroughly assisted by Dr. Crichton-Browne, who aided him in many ways, especially in supplying and describing pathologic specimens, besides fully and unselfishly contributing to his undertakings. Allbutt had had unusual opportunities at the hospitals for patients with mental disorders in the other "Ridings" in Yorkshire. So constantly was he encouraged in his studies by Jackson that he dedicated the book to him.

In the meantime the great West Riding Hospital for the Insane at Wakefield was thrown open to Dr. Allbutt. It is difficult to conceive that a more magnificent field for the study of cerebral disease could be found. Every form of the diseases to be observed was continually represented there; as many as 1,200 patients were then under Dr. Crichton-Browne's care. That generous and enthusiastic superintendent permitted the students of the Leeds School of Medicine to avail themselves of those great advantages.

In the second half of 1867 Allbutt undertook the ophthalmoscopic study of the patients at the West Riding Hospital for the Insane under the direction of Dr. Crichton-Browne, and at the North and East Riding hospital for the insane at Clifton, near York, under Dr. Christie, and he was greatly aided by these physicians. The conditions had been diagnosed and the cases selected for him; many were observed repeatedly, even three times. So numerous were the patients and so varied their conditions that he could not refrain from regarding a hospital for persons with mental disorders as a "museum of cerebral disease." In some instances abnormal states of the nerves were found; in others, a normal state. For convenience the conditions were classified as "general paralysis" (dementia paralytica), idiocy, dementia, mania, monomania and melancholia. The cases of insanity depending on a connection with epilepsy were regarded separately.

Allbutt had been led to inquire into the phenomena of insanity from a general interest with cerebrospinal disease; his primary object was the hope and expectation of finding in the retinas of patients with dementia

paralytica changes of or about the blood vessels such as several observers had described in the brain in persons who had died of that disease. Although he was disappointed, yet he was led to the discovery of a certain condition of the optic nerve which will be described later, and he determined to continue his examinations with the hope of finding more definite knowledge of various pathologic conditions than was appreciated in the study of nervous disease at that time, relating to a sorting out of the organic from the functional changes. Also, he expected to find from the study of the eyegrounds in the course of mania some indication of the state of the cerebral parts of the eye as related to the state of the cerebral circulation.

Allbutt put no reliance in statements concerning the visual powers. He regarded the failure of sight as estimated by the ability to read test types as "an oculist's symptom"; the results bore but little proportion to the amount of the atrophic or other diseases seen in the nerve. The mapping of the field of vision, however, he declared to be " a physician's symptom" and to be of the utmost importance in some cases. Both these tests were out of the question with patients with mental disorders.

Ophthalmic surgeons then, as nowadays, were seldom called on to examine a nerve in the early stages of the neuritis, because at these stages there was not noticed marked loss of vision. In the incipient stages atrophy can seldom be surely ascertained; its chief significance, therefore, lies in the important pathologic changes which have taken place. Allbutt regarded this process as chronic optic neuritis. He found that atrophy bore no fixed proportion to the ataxia of the orbital muscles that one sees in cases of dementia paralytica, but usually the changes in the nerves were proportionate to the contraction and dilatation of the pupils. In the early hyperemic stage the pupils are contracted, but they become dilated as white atrophy succeeds.

He scheduled 53 cases of dementia paralytica, in which he found no changes in the optic nerve and retina. In 48 others he found atrophy of the optic disk in various stages, and 7 he marked as doubtful. He came to the following conclusions: In almost every case of dementia paralytica at about the end of the first stage there were ophthalmoscopic evidences of atrophy of the optic nerves, which he believed traveled slowly from the centers and along the optic tract. Later, after he had made a large number of sections of the tracts and the corpora quadrigemina, he was forced to reverse his concluscion. At first the disks were hyperemic, with a slight exudation on their surface and without much stasis. Later these signs became white and staring, the edges of the disks being sharply defined. In other cases the exudation might be decided and in the end appear as though punched out.

He noted 51 cases of mania. In 25 cases, a surprisingly large number, the state of the nerves denoted symptoms of intracranial

disease. In many others permanent changes in or near the disk were noted. In the cases of mania dependent on organic as well as functional causes, within a few days after a paroxysm the back of the eye exhibited vascular suffusion, so pink in certain cases as to obscure the disk. During the paroxysm, however, the disk was anemic, perhaps from spasm of the vessels. Exudation appeared only when the damage was permanent, as from obstruction to the intracranial circulation followed by atrophy. The frequency with which hyperemia of the disk and retina were observed after a paroxysm led Allbutt to conceive the idea that he could detect cases of mania by that appearance only. In certain cases the conjunctiva became hyperemic, owing doubtless to paralysis of the vasomotor nerves. He imagined that the anemia of the retina and the whitish state of the disk, a probable atrophy, might indicate that the eye participated in a disturbance in the circulation of the brain whereby a spasm of the arteries produced epilepsy of the mental functions followed by paralytic dilatation of variable duration.

There were 48 cases of dementia not connected with epilepsy, in most of which the condition was severe, being due to organic disease of the brain. In 23 he found disease of the optic nerve and retina.

In a large proportion of the cases of melancholia and monomania the condition was functional only. Among 17 patients, the retina and optic nerve were healthy in 10. One patient, who later became ataxic, presented signs of commencing atrophy of the disk. The ataxia confirmed Allbutt's opinion that the signs in the disk were in their early stage. He frequently noted the presence of anemia of the retina in patients with melancholia.

Forty-three cases of insanity depending on epilepsy were studied, but in comparison with the other classes the findings were few and of no distinctive importance. Epilepsy alone, that is, when it is not dependent on organic disease, was not usually accompanied by disease of the optic nerve. Organic disease, however, was indicated by signs in the optic nerve unilaterally. The class of patients with this condition, and all the other patients were given repeated examinations, and the diagnoses were confirmed by several independent observers.

Among the idiots, atrophy of the optic nerve was not uncommon. Among 12 patients, 5 presented decided atrophy, and another showed advancing disease. Whether this condition had been caused by encephalic inflammation in childhood Allbutt left for future observers to decide.

The main interests of Allbutt's communication lie in the schedules that he so perfectly prepared to show the details of his examination of 219 persons. In a large proportion of cases of old or severe organic disease of the brain and its membranes, whether degenerative, hemorrhagic, meningitic or due to tumor, changes in the eye were seen. In

the cases of so-called functional disease such changes were found in a few instances. At that early day of scientific neurology the occurrence of atrophy of the disk as that condition is now seen, as a constant symptom in cases of dementia paralytica, became a fact of the greatest pathologic importance. In cases of functional mania and functional epilepsy the variations of vascular tension in the retina and disk suggested to Allbutt and his colleagues fields of observation for later investigation.

Allbutt was persistent and most searching in his examinations and employed every available method and maneuver. He made repeated examinations in any state of the optic disks, pursuing his examination whenever possible, even during the course of an epileptic seizure or in coma or during a maniacal attack. His testimony settled for all time the fear of those timid physicians who refrained from using the ophthalmoscope lest the eye of the patient might be damaged by the heat and glare from the mirror. He did not fear to use a strong light, despite Jaeger's warning that strong lights might be dangerous.

Allbutt's studies led him to certain conclusions as to the value of the ophthalmoscopic signs in the diagnosis concerning cerebral diseases; he noted especially that in nearly every case of dementia paralytica atrophy of the optic nerves may be observed throughout the whole course. Allbutt was the first, in England at least, to make this observation. It is true that Westphal, of Berlin, with von Graefe's assistance, was similarly engaged; his researches were far less complete than Allbutt's, yet his conclusions were the same.[9] These findings were not then considered to be of special diagnostic value, yet they were of great pathologic significance as throwing light on the minute changes of structure which cause the more characteristic symptoms of that disease.

These studies yielded the investigator much about which he had had no previous knowledge; his honesty compelled him to make inquiries as to what observers in other countries had seen and noted. When Allbutt began his studies he was ignorant of the German language; to further his investigation he learned German and was able to know what von Graefe and others were accomplishing in their labors in the same field. He therefore was greatly encouraged on learning the results of their independent records.

The lectures on optic neuritis formed part of a course in clinical medicine delivered at the Leeds School of Medicine. The lectures were necessarily not exhaustive, as they were designed for practitioners and senior students. Allbutt, after giving a short sketch of the anatomy of

9. Westphal, C. F. O., in Allbutt, C.: On the Use of the Ophthalmoscope in Diseases of the Nervous System and of the Kidneys, New York, Macmillan & Co., 1871, p. 193.

the parts of the eye with their connections and origin, described the vascular parts of the optic nerve and retina and their connection with the brain. Then he gave a short explanation of the ophthalmoscope, at that time employed by him by the indirect method which produced the reversed image of the back of the eye. After a brief account of the fundus, the details of which were obtained only after full mydriasis induced by atropine, the lecturer pointed out that the retina was not the expansion of the fibers of the optic nerve but rather a structure distinct from the optic nerve, possessing a separate system of nutrition. Despite the fact that they are antiquated, the lectures are of great interest even today and are as vital as when they were delivered. They show how the knowledge had developed from the first views ever obtained of the ocular fundus up to the days when the gross details were made visible by instruments vastly more efficient than Helmholtz' original invention and had become so acceptable in their definition as to justify the preparation of an atlas, such as Liebreich's, to serve as a guide for all observers of the fundus. By this time it had become apparent that the eye was an outpost of the brain. Though the minute details of the retina and optic nerve had not yet been seen, sufficient was known of the apparent small details to justify the assumption that the morbid changes seen were not confined to the ocular structures but were an extension of what was presumed to be existing within the cranium, made visible by the ophthalmoscopic mirror, so that the changes of "optic neuritis" could be described in detail.

It might be of interest to those who today are teachers in medical schools to read the advertisement which was put out by the Leeds School of Medicine. In the announcement for the winter session, to commence on Thursday, Oct. 1, 1868, Dr. Allbutt is scheduled for demonstrations in clinical medicine and for a course in the "Principles and Practice of Physic." And for the summer session of 1869, commencing on May 1, he is listed as giving a course in comparative anatomy. The clinical lectures were given at the Leeds General Infirmary. The fees for attending the course in medical practice were, for the winter session, £ 7, 7s.; for the summer session, £ 6, 6s.; for twelve months, £ 12, 12s.; for eighteen months, £ 15, 15s.; for three years, £ 21, and in perpetuity, £26, 5s.

In January 1868 Allbutt [10] presented an analytic and critical review of a number of papers by various authors published in England and France from 1860 to 1867 on subjects connected with the ophthalmoscopic study of alterations of the optic nerve and of cerebral diseases on which such alterations depend. At that time knowledge of the

10. Allbutt, Thomas Clifford: Brit. & For. Med.-Chir. Rev. **41**:127 (Jan.) 1868.

diseases of the cerebral nervous system was meager, largely because of the lack of method in their investigation. The functions of the brain and the complexity of the phenomena as they were then understood gave rise to theories incapable of being tested by experiment and unchecked by reference to nature. The application of the ophthalmoscope, an instrument devised for the study of the eye and its particular structures, to the diagnosis of nervous disorders, however, gave one an insight into the necessity for the utilization of other instruments of precision which at that time were crude and inefficient. Already, Ogle, Allbutt and Jackson, and Carter and Hutchinson, in England; Galezowski and Bouchut, in France, and von Graefe and Saemisch, and Schweigger and Sichel, in Germany, were using the ophthalmoscope and appreciating the marvelous changes which the instrument had produced in the knowledge and method of the oculist.

In the review Allbutt presented a systematic yet brief description of the ophthalmoscope, the methods of using it and the details of the ocular fundus, with the straightforward simplicity and clearness which characterized all his writings. In this account were revealed the signs of the diseases of the optic nerve and retina which he observed in the analysis of the cases that he had studied at Wakefield hospital for the insane. His article must have made a strong appeal to the cultured general practitioners of medicine and surgery familiar with the English "Quarterlies," who might not have been attracted to the occasional report in the weekly professional journal.

The essayist unhesitatingly declared that not only did the ophthalmoscope clear up many doubts and enable one to recognize certain pathologic states which before were beyond reach, but it encouraged new habits of accuracy, which habits became reflected in the work of other departments of practice where the ophthalmoscope was less needed. The ophthalmoscope helped the science and practice of medicine not only by the facts which it directly revealed but stimulated work in the direction of nervous diseases especially, for by means of the ophthalmoscope one was enabled to see for the first time in the history of medicine the commencement and progress of changes in the life of nervous tissue and to ascertain the modes and times of such changes. Through the teaching of those whose works were referred to it became clear that the instrument should have been in the hands of every physician who wished to speak with authority on the subject of diseases of the nervous system.

In the perusal of these pages written nearly seventy years ago one must bear in mind that at that day the ophthalmic practitioner was not accorded the place in the estimation of the general practitioner which was ascribed to him twenty years later. The review said just enough to show that various and important indications of disease were to be

found in the eye and that physicians were in possession of a vast number of facts which helped in the diagnosis of many hitherto obscure diseases and marked states of tissue.

By this time Liebreich's atlas of the fundus had been published. This contained admirable plates to show the chief diseases of the retina and optic nerve. So important were the facts now available that it became clear that they no longer could be neglected on the score of their minuteness and that instruction in ophthalmic practice should be given to both medical and surgical students. The review closed with quotations from Sir Thomas Watson, who through the influence of Allbutt's persistence began his course of lectures in medicine with four lectures on diseases of the eye and who declared that "we find in the eye more satisfactory and plain illustrations of the general facts and doctrines of pathology than in any other part of the body." And, to use the words of Dr. Latham, "Here you see almost all diseases in miniature, and you learn many of the little wonderful details in nature of the marked processes which but for the observation of them in the eye could not have been known at all."

Looking back at the events of 1865 to 1875, one knows that the words of Sir Thomas Watson and Dr. Latham daily gained weight and a meaning which previously could be no more than guessed at by their distinguished authors. The fruits of those workers settled for all time the truth of the assumption that damage or a change of state in the eye is not uncommonly coincident with nervous and other diseases. Even at this early day in his career Allbutt's influence became of prodigious importance, and as physicians became aware of the facts which he pointed out as necessary to be observed wherein he sought to strengthen inference into certainty they worked on. Yet they could attain no thorough knowledge of the significance of optic changes in association with cerebral disease until they had become familiar with the modes of the production of those changes. Between those concurrent events there lay a chain of causation which turned out to be a series of phenomena of infinite value, as amply attested by many cases of high interest.

In commenting on Dr. Hughlings Jackson's aphorism that "optic neuritis depends upon coarse intracranial disease," Allbutt stated the belief that such a doctrine does not apply to "chronic neuritis," and he inquired as to the value of that aphorism, not because he doubted the truth of Jackson's statement but because he wished to know what that scrupulous and thoughtful observer implied therein. Allbutt ventured to say that "Dr. Jackson was inclined to use words 'loosely.'" However, he did not imply that there was looseness in the thought but rather that, owing to the deficiencies in the contemporary knowledge, Jackson

was compelled to coin his own phrases for the meaning of which he alone became responsible among those who dare to employ such phrases. Allbutt declared: "They like loose words, therefore, not that they may easily slip them on, but that they may easily slip them off, so that there may be room for their thoughts to move at ease within them. Unluckily, however, there are many other people who gladly catch at any expressions that seem at once fine and easy." For this reason Allbutt was disposed to deprecate the use of such an aphorism as that of Dr. Jackson about optic neuritis and coarse disease, just as he deprecated the latter's use of the term "cerebral fever," which term fits too many conditions too easily and encourages in others the very mental indolence prevented in Jackson. In the series of 200 cases which Allbutt presented before the Royal Medical and Chirurgical Society he sought to show that optic changes were chiefly or entirely coincident with static changes in the brain centers, yet he hesitated to speak of those changes as indicative of "coarse" disease. However, he would call a tumor pressing on the cavernous sinus or severing the connection between the optic nerves and their ganglions coarse disease, but would not so denominate the belt of cerebritis around it. Dr. Jackson was inclined to regard a small abscess as coarse disease, but Allbutt would not consider a small spot of softening coarse. A rude clot may be far coarser than a small patch of meningitis about the chiasma, but the clot leaves the eye uninjured, while the meningitis sets up optic neuritis. Dr. Jackson would hesitate to say, "The coarser the disease the more the neuritis," and yet in Allbutt's opinion it should be so if the coarseness were a direct element in its causation. A clot, he would say, though coarse disease is not itself an encroaching disease; so he would propose a change in his friend's rule by saying that optic neuritis depends on encroaching disease, whether coarse or not, including not only ischemia and acute neuritis, but chronic neuritis also.

In the series of lectures Allbutt confined himself to the most obvious details and refrained from debatable subjects. Through all his lectures and at their conclusion he testified to the thoroughness and the attractiveness of the able papers of Hughlings Jackson.

The effects of his entire course of study of the cases referred to in these pages only encouraged Allbutt to maintain what he and Hughlings Jackson declared from the beginning of their careers, that the "account of a cerebral disease cannot be complete without the record of ophthalmic observations."

To serve as a general doctrine concerning optic neuritis, he ventured to pronounce certain aphorisms, such as: "Ischaemia is always due to pressure; Neuritis to encephalitis, cerebral or meningitic." In cases of tumor in which there were ischemia and neuritis also, Allbutt was not always able to satisfy himself as to the etiology, because the pathologic

features were not yet definitely understood at that date, although he was inclined to regard the tumors as ischemic without disease of the optic nerve trunks. Few writers in that decade used the ophthalmoscope in cases of cerebral disease. Allbutt determinedly sought for autopsies and performed them whenever possible; in numerous cases he observed inflammatory changes in the brain substance surrounding the neoplastic or cystic body and on the meningeal coverings. He became aware that such augmentations in the cerebral contents excited an increase in the intracranial pressure, which extended its force into the foramina in the basal fossae. Many physicians at that date held that ophthalmoscopic research could not reveal anything of the nature of a cerebral tumor; nevertheless, Allbutt sought every opportunity to examine and reexamine repeatedly the fundi of those suspected of having intracranial disease. He regarded the occurrence of optic changes as not only the one symptom among many but often the one important symptom among a few. He did not hesitate to decide on the approximate location of a tumor but stated that tumors of the base of the brain commonly implicated the optic tract, and, if present in the anterior fossa, they eventually would press on the cavernous sinus, the chiasma or the tract; tumors of the posterior fossa seldom interfered with the optic disks, but, when very large, they might have compressed the medulla and extended to the quadrigeminal bodies or were propagated to the cerebellar peduncle. Tumors of the cerebral lobes, he found, affected the optic nerves variously. Allbutt was compelled to rely on his own experience in presenting the accounts of their effects; he believed that optic signs sooner or later appeared in almost all cases of tumor in the anterior lobes and in the middle fossa, yet he had found complete bilateral atrophy in cases of tumor of the posterior lobes.

During his observations Allbutt was impressed by the truth of the fact which Hughlings Jackson had repeatedly remarked and which was insisted on by von Graefe, that although the optic disk and its neighborhood might be greatly changed central vision can still be normal. Another clinical item was to the effect that greatly swollen disks can become reduced and the resulting vision higher than that in cases of descending neuritis, in which latter condition the visible contour of the disk might be but little changed.

Again, Allbutt, like Jackson, regarded all statements as to visual powers as useless; in all instances he endeavored to map the visual field. The notes of his experience tell of the subjective and objective symptoms following involvement of the sections within the various fossae of the base of the cerebrum. He was well aware that disease of the

cerebellum resulting from pressure and from inflammatory change may cause amaurosis because of the nearness of the quadrigeminal bodies. Galezowski and other continental observers were inclined to declare for inflammatory processes, but Allbutt maintained that in the greater number of cases the condition was caused by tumor. Hemorrhage into the optic centers was invariably followed by amaurosis, though the effects on the nerve were not always shown early by ophthalmoscopic signs. The keenness of Allbutt's interest in the subject was manifested in the thoroughness with which he pursued his study of cases. He watched the results of all the cerebellar tumors in the patients under his care and noted their relations to the quadrigemini. He examined microscopically the tissues around the tumor, as well as the peduncles and the quadrigeminal bodies themselves. He remarked that acute diseases, such as cerebral hemorrhages, when occurring alone never caused ischemia or neuritis. A clot, as a rule, did not enlarge and arrest the movements or force of the blood but became contracted and consequently withdrawn into itself. Allbutt's belief was that "it is not a sudden thrust but a gradual invasion which does mischief." Yet clots were often antecedents or sequences of softening and were often associated with neuritis, generally with chronic neuritis or simple atrophy. Allbutt agreed with that most acute and trustworthy observer, von Graefe, but differed from Galezowski and others who rarely observed optic signs in cases of cerebral hemorrhage, in noting that hemorrhage in the region of the thalamus might exert pressure on the tract, with resulting hemiopia.

Allbutt had opportunities to observe from its commencement the process known at that early ophthalmoscopic day as "simple white atrophy," a condition observed by the ophthalmic surgeon then, as today, generally only when loss of sight had set in and the atrophic process was almost over.

As a name for certain lesser degrees of resistance to destruction which often precedes white atrophy Allbutt introduced the term "red softening of the optic nerve," by which he meant the presence of only slight congestive appearances, accompanied with but little disturbance of the cerebral vessels. When patients with the early stage of cerebrospinal disease were examined he usually found many presenting the signs of so-called simple progressive atrophy, which showed a distinctly hyperemic stage, with little effusion preceding the stage of whitening. This condition was not noted in the textbooks on diseases of the eye of his day, but to Allbutt's great delight he found that it had been

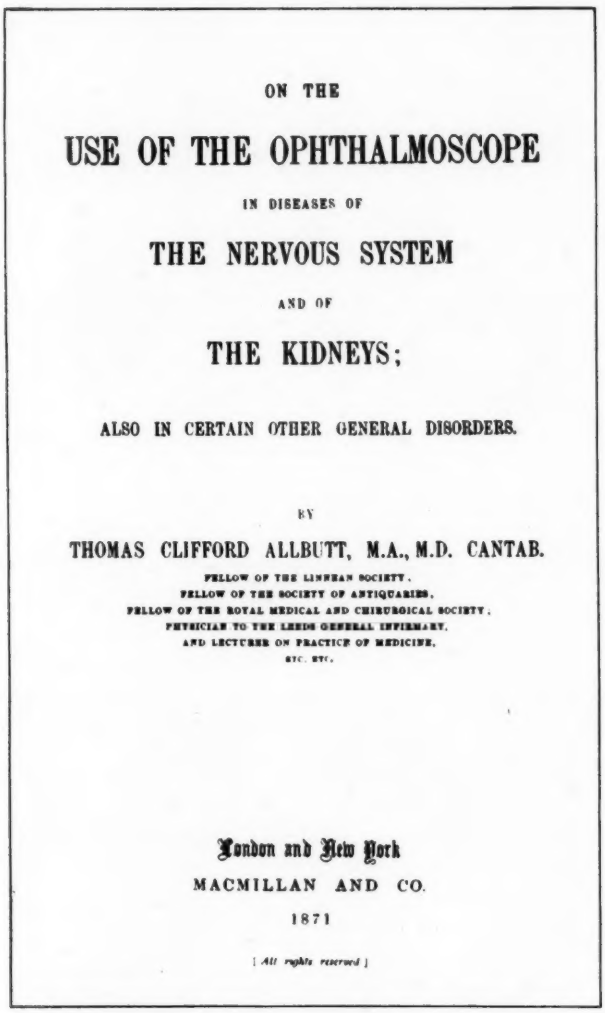
described in 1864 by Jonathan Hutchinson.[11] In a microscopic study of a case he found excessive fibrillation but no marked proliferation of the nuclei. Allbutt was not able to determine the pathologic cause of this condition; he found chronic optic neuritis in cases of dementia paralytica and locomotor ataxia. Allbutt had had many opportunities for postmortem examinations, an unusual privilege at that time in England. He observed frequently in advanced cases of dementia paralytica alterations in the optic papillae and nerves, in the tracts and in the quadrigeminal bodies, although in recent cases the corpora quadrigemina were not deeply affected. He did not find such changes in the cases of ataxia, however. At that time to Allbutt and to all others working on the subject the cause of chronic neuritis was obscure; certainly it could not be explained by Hughlings Jackson's idea that "optic neuritis depends upon coarse disease." Allbutt ascribed it to "encroaching disease whether coarse or not." When white atrophy was seen for the first time he did not attempt to ascribe it to any particular precedent process, nor did he try to distinguish the acute from the more degenerative form of neuritis. It was generally noticed that the two eyes were unequally affected, a fact which offered some help in the diagnosis. Complete atrophy was found to be present a year or two before decided symptoms of disease of the brain, such as paralysis, betrayed the real seat of the disease. In his teaching Allbutt insisted that the early symptoms should always be noted. The pupillary actions might vary; indeed, not always were the pupils dilated or even inactive. But the field of vision was always lessened laterally before central vision was much disturbed. Occasionally he found only one eye atrophic, and in some cases of blindness the disks presented no visible changes.

The monograph entitled "On the Use of the Ophthalmoscope in Diseases of the Nervous System and of the Kidneys; Also in Certain Other General Disorders," which Allbutt published in 1871, received general attention, as it well deserved, not only because it was the work of a highly intelligent and eminently scientific physician but because Allbutt had devoted his great abilities to the elucidation of what at that time was a literally dark subject. The use of the ophthalmoscope in ordinary medicine had been so recently adopted that only those physicians who had striven to use the ophthalmoscope systematically in their ordinary work could tell how difficult it had been to put the right interpretation on what they had observed. There was no book to which they could refer to aid them in their difficulties. The book was especially

11. Hutchinson, Jonathan: Clinical Data Respecting Cerebral Amaurosis, London Hosp. Rep. **1**:33, 1864.

welcome to such physicians, while many to whom the ophthalmoscope had been an unknown instrument were encouraged to take it up and work with it since its value had been made clear to them.

Allbutt began with an account of the introduction of the ophthalmoscope into ophthalmic practice and its gradual application to the



ON THE

USE OF THE OPHTHALMOSCOPE

IN DISEASES OF

THE NERVOUS SYSTEM

AND OF

THE KIDNEYS;

ALSO IN CERTAIN OTHER GENERAL DISORDERS.

BY

THOMAS CLIFFORD ALLBUTT, M.A., M.D. CANTAB.

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ETC. ETC.

London and New York
MACMILLAN AND CO.
1871

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Fig. 9.—Title page of Allbutt's monograph.

elucidation of other than purely ophthalmic problems. Next he detailed the mode of using the instrument. For general use he employed a metallic mirror and two object glasses. He condemned all fixed apparatus as useless and cumbersome. He advised physicians "while learning to handle the apparatus to operate on an eye whose pupil had been

dilated by atropine; yet the sooner dilatation could be dispensed with the better, for patients did not like it, as the disordered accommodation remained long after the necessary examination, and normal vision was consequently interfered with." He assured all that they "might speedily get accustomed to dispense with the use of atropine." The succeeding chapter is devoted to anatomic details, the most important of which at that time seemed to have been that the vascular supply of the optic disks was connected with the brain system rather than with the retina and that the coverings of the optic nerve consisted in an actual prolongation of the arachnoid interspaces.

The anomalies of the optic disk, which are puzzling to the beginner, were next considered. Those alluded to were the varied distribution of the retinal vessels, persistence of the hyaloid artery, pulsation of the veins of the retina, the physiologic excavation of the disk similar to that existing in glaucoma, and the troublesome anomalies in the color of the disk, having an important bearing on the significance of hyperemia and anemia, as only the experienced observer could say what was physiologic or natural and what was pathologic or the result of change due to disease. Edema and ischemia were pointed out as being equally annoying, for the latter could be confounded with inflammation, commonly under the perplexing term optic neuritis. To differentiate the truly inflammatory process the term "neuroretinitis" was introduced, while the term "ischaemia" was applied to conditions superficially somewhat similar but due to vascular arrest.

The term "chronic optic neuritis" was retained to indicate the subacute inflammation which so commonly preceded white atrophy. "Retinitis" and "perineuritis" were pointed out to be of great importance medically, as in daily practice the diagnosis between "consecutive atrophy" and "primary atrophy" is not always easy.

The relations of these conditions, so lucidly described, to intracranial disorders were next considered, especially those cranial disorders involved in epilepsy, chorea, mania, dementia, meningitis, concussions and fracture, hydrocephalus, tumors and periostitis chronica, atheroma, softening and hemorrhage of the brain, cerebritis, abscess and sclerosis and, finally, dementia paralytica. Allbutt had noted that during the seizures of epilepsy the disk was somewhat vascular, the vessels being generally larger and fuller than usual, while in chorea no change was seen, as a rule. In acute mania, just after a paroxysm there was slight hyperemia, or pinkness. Many changes, but none very marked, were seen in dementia. The ophthalmoscope enables one to come to an early diagnosis in cases of the tuberculous form of meningitis. Long before other symptoms are visible, congestion of the optic papillae may give

warning of the approaching danger. Ischemia could be seen in the early stage of hydrocephalus, to be followed later by simple white atrophy. Much had been accomplished by the ophthalmoscope in connection with intracranial tumors. In dementia paralytica of persons with mental disorders observers had detected a tendency to simple white atrophy of the disks. Disease and injuries of the spinal cord often alter the optic disk, thus manifesting a connection between the two, but in 1870 much remained to be done before the ophthalmoscope could be relied on in the discrimination. The morbid conditions of the eye accompanying albuminuria, then spoken of as "albuminuric retinitis," afforded an opportunity for the ophthalmoscopist to draw valuable conclusions as to the existence or nonexistence of that malady. As the disease may exist in a marked degree without the appearance of dropsy, that fact is of sufficient importance to impel physicians to cultivate the use of the ophthalmoscope. And such might be the case in leukemic retinitis, with its peculiar and characteristic symptoms. Toxic amaurosis was touched on, and the classic story of embolism of the arteria centralis was mentioned. The book made no pretense to present a complete history of the morbid conditions of the retina in all kinds of disease; at that time no such compilation existed. The work served as an impartial review of the literature and was at once recognized as "full of sound reasoning based on honest observation, never carrying conclusions further than the available data justified, and assembling all the known facts about the subject" and "not entertaining," as the author quoted from Locke's 'Human Understanding,' "any proposition with greater assurance than the proof well warrants."

Dr. Ogle,[12] who must have taken great pride in having directed Allbutt to take up ophthalmoscopy, wrote as follows of the monograph on the ophthalmoscope:

> The great merit of the work is, that it clears the way for other workers. By its aid men will no longer be compelled to work for years in the dark—they will have a definite standpoint whence to proceed on their course of observation. And we think it well to say a word on the spirit of the book, which is to us most pleasing. It is not a book of controversy; in it all fellow-workers are spoken of in the same loving spirit. From the kindly dedication to Dr. Hughlings Jackson to its "Finis," this treatise has only words of encouragement to those workers for whom the author lays bare all his stores—the accumulations of much hard work both in the field of observation and in that of research. Did it not seem unkind, we should be tempted to desire that a new edition might be deferred until these stores had fructified and produced the ample harvest of exact knowledge the labour spent in their acquisition deserves.

This important piece of scientific and literary work is exceptional in that, contrary to Allbutt's usual practice in later years, he never

12. Ogle, John W.: Brit. & For. Med.-Chir. Rev. **49**:427, 1872.

brought out a second edition or returned at length to the subject. But he had hoped to follow up the volume by another essay on the disturbances of motion and nutrition of the eyeball. In a personal note to me written in November 1924, Sir Clifford, in response to an inquiry that I had made of him, referred to his monograph on the ophthalmoscope as "sere and yellow."

It is interesting to know that Allbutt for a time used the long tube ophthalmoscope of Lionel Beale, carrying the mirror, the lens and the lamp, such as that used by Hughlings Jackson. But he abandoned it because he "did not like to change the lenses at will, or to pass from the indirect to the direct examination, all of which is impossible in fixed instruments." He seems to have preferred a metal concave mirror of about 10 or 12 inches (25.4 or 30.5 cm.) focus, with a clip behind it to hold ocular lenses, much like the type devised by Brudenell Carter, with two convex object glasses, and a concave glass for the direct examinations. Much of this study was carried on by the indirect method; occasionally he employed a binocular or a stereoscopic instrument.

Notwithstanding the advice of Hughlings Jackson that he should work at medical ophthalmology, Allbutt did not enter ophthalmic practice but used his ophthalmologic knowledge to aid him in his general interest in clinical medicine. The disorders of the circulation which Trousseau had led him to observe particularly attracted and maintained his attention for many years, indeed, as long as he lived.

After comparative silence for a number of months Allbutt[13] published a decidedly critical paper on the cause and significance of choked disk in intracranial disease. It must have been gratifying to him to see the fruits of his labors and to note that practitioners in England, France and Germany could not dispense with the use of the ophthalmoscope in the study of nervous disease. The headaches complained of by a syphilitic patient could not be understood nor the "biliousness" manifested in the course of an intracranial tumor explained without an ophthalmoscopic examination. In the case of syphilis the optic nerve might be inflamed or atrophic, while the effects of tumor might lead to congestion of the papilla, producing the *Stauungpapille* of von Graefe, or the "choked disc" of Allbutt. Through a natural increase in his knowledge since the preparation of his earlier papers and the monograph the author sat in judgment on his own contribution and that of other observers on the continent. He made a sharp distinction between optic neuritis, implying a true inflammation, descending through the course of the nerve, and choked disk. In the ten years of his experience he frequently encountered cases in which the elements were mixed, the diagnosis becoming therefore difficult to decide.

13. Allbutt, Thomas Clifford: On the Causation and Significance of the Choked Disc in Intracranial Disease, Brit. M. J. **1**:443, 1872.

Few investigations in medical science and practice in the decade of 1865-1875 excited more interest than those made into the connection of the changes in the eye in association with diseases of the central nervous system. The various hypotheses advanced to account for the causation of choked disk both clarified and confused the pathologic conceptions held by all observers. Allbutt endeavored to present the

D'C. E. Fitzgerald ad nat pinx. Morison & C° Imp. Dublin

CONGESTION PAPILLA
Enlarged 15 Diameters

Fig. 10.—Frontispiece of Allbutt's monograph.

latest developments of the problems in his monograph. In his later and final paper on ophthalmologic subjects he referred to the investigations of Manz, Schwalbe, Schmidt, Axel Key and others. His theory that *Stauungpapille,* or choked disk, depends on excessive intracranial pressure coincided with that held by von Graefe. At that time he was

uncertain as to the mode in which the pressure makes itself felt. In effect he strongly believed that the intracranial pressure forced the fluids into the neural sheaths and that, owing to dropsy, the vaginal spaces pressed on the nerve and caused the swelling, the choking of the disk arising from the retention of the fluid in the nerve head by the narrow scleral ring. There seems to have been in most cases a definite relation between the amount of intracranial fluid and the dropsy of the nerve. As to the association of descending neuroretinitis and choked disk, Allbutt stated that he did not remember ever seeing a well marked swollen disk pass into the typical form of neuroretinitis. In conclusion, he had found no hypothesis which fully demonstrated the causation of descending neuritis.

In addition to reporting his own investigations in his chosen subjects, Allbutt occasionally wrote reviews of books and biographic notices of distinguished persons. There were two brief articles of interest to the ophthalmologist. The first is a notice of Galezowski's [14] article on the diagnosis of diseases of the eye by means of retinal chromatoscopy. Galezowski, who for years had been interested in color blindness, a condition which was for a long time in France cruelly spoken of as *daltonisme*, pointed out in his book that color blindness might be acquired as the result of certain conditions of the optic disk and retina and that colors might be employed in the tests for ascertaining the condition of the optic nerves. The second article of ophthalmologic interest was a sympathetic and generous appreciation of the life and works of Jules Sichel, who died toward the end of 1868. When one knows the characteristics of Allbutt one can see how readily he would appreciate the personal greatness of Sichel, who did so much to place diseases of the eyes on a scientific basis.[15]

For a number of years Allbutt occupied the chair of "Principles and Practice of Physic" and of "Materia Medica and Therapeutics" in the Leeds School of Medicine and was in charge of the museum, a fitting reminder of his boyhood interests. When he was made consulting physician to the Leeds General Infirmary in 1884, he resigned most of his lectureships but continued to be actively interested in the medical school. As president of the council he effected notable reforms over which he had control, in the system of medical education, and in time the medical school became a part of the University of Leeds. In his clinical lectures he adopted the methods of Trousseau, whom he much resembled. In his interpretations of the ideas of others he manifested

14. Allbutt, Thomas Clifford: Du diagnostic des maladies des yeux par la chromatoscopie rétinienne précédé d'lune étude sur les lois physiologiques des coulours, by X. Galezowski (book review), M. Times & Gaz. **24**:490 (Oct.) 1868.

15. Allbutt, T. C.: M. Times & Gaz. **2**:676, 1868.

in a charming manner a singular clarity of exposition, which made a strong appeal to all the students, thus increasing his popularity with them.

He sought to lay practical stress on the true method of investigating disease. He endeavored to discover the minute and early deviations from health rather than to be concerned only with great examples of damage. He considered it one's duty to familiarize oneself with the general laws and modes of the growth of tissue as seen grossly or microscopically and also to learn the special modes of the growth of tissue for each particular part.

By his invention, in 1867, of a short graduated thermometer tube which could be employed readily on the person, the use of which became established as a routine procedure, Allbutt should be given credit for the introduction of thermometry into clinical medicine. One of the fruits of his interest in thermometry was a paper on "The Effects of Exercise on the Bodily Temperature," which he read before the Royal Society in the same year that his work on the ophthalmoscope appeared. And another paper, on the significance of cutaneous diseases in the classification of diseases,[16] was published in 1867. In the next year, he published in St. George's Hospital Report, a short-lived periodical, an epoch-making paper of much importance to the ophthalmologist on the histologic changes in syphilitic disease of the cerebral arteries.[17] It commonly has been asserted that Huebner was the first to make the observations reported, yet as Huebner's report was not published till 1874 the credit should be given to Allbutt for the discovery of this condition of the brain.

Disorders of the circulation particularly attracted Allbutt's attention. He introduced the terms "senile plethora" and "hyperpiesia" to describe the rise of the arterial blood pressure that occurs in otherwise healthy persons after middle life, especially noting the rise of tension in men who had discontinued athletic habits but continued to indulge in an abundance of food.

Among his contributions to the progress of medicine were special studies of the arthritic symptoms and the visceral neuroses in association with locomotor ataxia. He accordingly chose as the topic for the "Goulstonian Lecture," which he delivered in 1884 before the Royal College of Physicians, "The Neuroses of the Viscera," a subject that was only then beginning to be understood.

The Goulstonian Lectureship was founded in 1632 by Dr. Goulston, who bequeathed to the Royal College of Physicians £200 for the main-

16. Allbutt, Thomas Clifford: The Significance of Skin Affections in the Classification of Diseases, St. George's Hosp. Rep. **2**:187-204, 1867.

17. Allbutt, Thomas Clifford: Histological Changes in Syphilitic Disease of the Cerebral Arteries, St. George's Hosp. Rep. **3**:55-65, 1868.

tenance of an annual lectureship "to be read by one of the four youngest doctors in physic of the College, between Michaelmas and Easter in three days together both forenoon and afternoon for an honorarium of £10." Thus, for more than two hundred and fifty years the Goulstonian Lectureship had been handed out as a form of compliment to the youngest of the fellows reckoned on the basis of their election. Allbutt was then 58 years of age!

Fig. 11.—Allbutt in about 1880.

Desiring to be relieved somewhat of the burden of his enormous and steadily increasing consultation practice, with a prospect of greater leisure in which he might devote himself to wider things of the medical life, in 1889 he accepted the post of Commissionership in Lunacy, an official government position. At that day the salary of the most highly remunerative public medical appointment in England was £1,500. His professional life in Leeds accordingly came to a close, and he removed to London, where he continued to reside until 1892, when he was elected Regius Professor of Physic at Cambridge, at half the amount of salary

of his commissionership. Allbutt then removed to Cambridge, where he continued to reside until his death. From the day of his election, though over 50 years of age and without experience as a professor, he took his place in the front of university life. This ancient professorship, founded in 1540, had been held by a distinguished line of physicians including, among other notables, Francis Glisson, the anatomist, physiologist and pathologist.

Allbutt, who had been a member of the Royal College of Physicians since 1878, was elected a fellow in 1883, at the same time as William Osler, who was then only 33 years of age! The Royal College of Physicians, perhaps the most ancient society of physicians in Europe, received its charter from Henry VIII in 1578, having been founded by the King's physicians, of whom Linacre was the leader. It was a singular coincidence that Allbutt and Osler should each have been destined to achieve the high honor of the Regius Professorship, the one in Cambridge, the other in Oxford.

During his long career in medicine, covering more than sixty-four years, Allbutt received practically every honor that may come to a British physician. In 1866 he became a fellow of the Royal Medical and Chirurgical Society of London and contributed papers to its transactions. Besides his membership in British and foreign societies, from which he received honorary degrees and numerous titles of distinction, in public life he was consulting physician to many institutions.

In 1880, for his original work in clinical medicine the Royal Society bestowed on him the honor of fellowship, and he later served as vice president.

On June 27, 1904, in Convocation, before a brilliant assemblage in the Sheldonian Theatre, Oxford, the degree of Doctor of Science was conferred on him, in company with Jonathan Hutchinson, Sir Patrick Manson, William Osler and others.

He was associated also with societies and clubs which gave him the right to affix to his name the insignia of honors which England bestowed on the greatest of her men. In 1907 he was created Knight Commander of the Bath, and in 1920, Privy Councillor, the only man to be called to the Council as a physician, and was frequently called into consultation notably as a member of government commissions, especially that of the Home Office Inquiry into Trade Diseases. He was also Justice of the Peace in two counties. During the World War he served on several important military commissions and was honorary Lieutenant-Colonel of the Royal Army Medical Corps.

In 1896, he began to issue his great "System of Medicine," which was completed in eight volumes in 1899. Many of the articles in this edition were contributed by Allbutt himself. In 1896 there also appeared his "System of Gynecology," embodying the most recent advances in

the field culled from the literature of the world. In 1915 he finished "Diseases of the Arteries and Angina Pectoris." In this great work he expressed the theory that angina pectoris is due to disease of the root of the aorta. At first the idea was not received with much favor, yet in time it steadily gained adherence and is now believed by all.

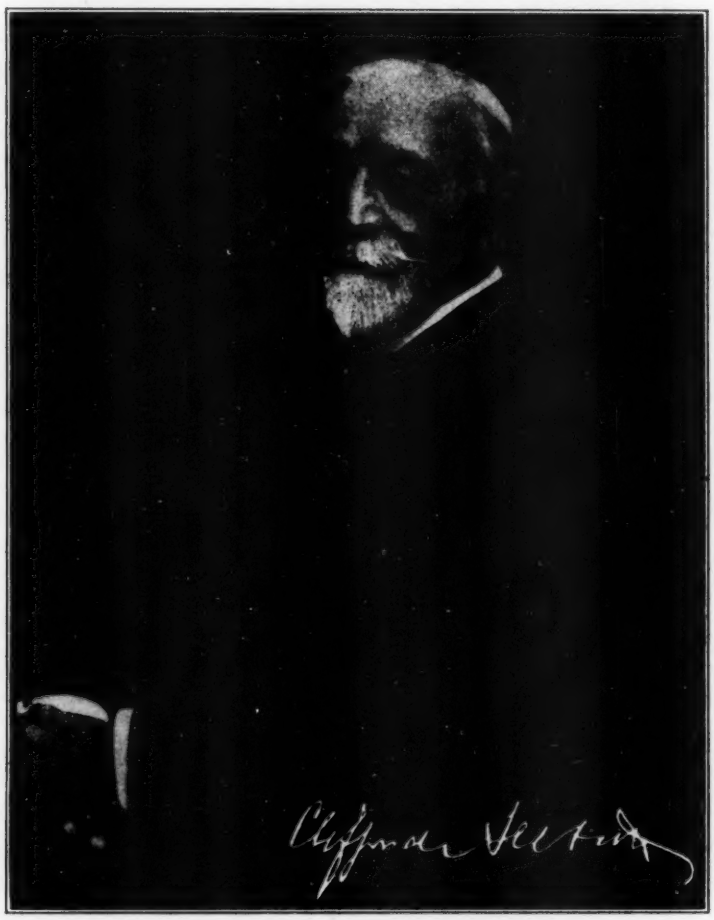

Fig. 12.—Allbutt in about 1899.

Another subject near to his heart which had for its object the consideration of the diseases of human beings and peoples and disease among animals was that of "Comparative Pathology," a subject which up to that time had been neglected, if not ignored. Allbutt urged that the manifestations of diseases and their effects on different individuals and races should be studied by comparison. He was so earnest an advocate of this extension of medical study that a section on this subject was formed in the Royal Society of Medicine, and Allbutt, appropri-

ately, was made the first president. Later he succeeded in establishing at Cambridge the now well known Institute of Comparative Pathology.

To the present generation Allbutt is best known, perhaps, as a medical writer with a pleasing yet powerful literary style. He was always more concerned with principles than with details. His scientific interests were varied; his contributions to literature all manifested the highest degree of scholarship. His knowledge of the literary side of medicine, both ancient and modern, was unrivaled and inexhaustible. Thinking and working along classical lines, like the true classical scholar that he was, he aptly and freely sprinkled quotations from Greek and Latin authors through the pages of his publications and in his correspondence.

His scholarship and wide knowledge of medical history served well to equip him to lecture on that subject. As a contributor to the literature on medical history he wrote papers and books on medieval science,[18] surgery,[19] Greek medicine in Rome[20] and Byzantine medicine.[21] In 1909-1910 he delivered a number of addresses under the title of the Fitzpatrick Lectures on the history of medicine, at the Royal College of Physicians. A reading of his contributions on the "History of Medicine" in the eleventh edition of the "Encyclopaedia Britannica" will serve to show the wide scope of his knowledge.

He strongly held that good diction and literary style are as desirable in science as in literature and insisted that the character of one's writing "should be as carefully attended to as he felt that he himself was bound to express." As Regius Professor he had to read a great number of theses; in many of these, while he found the matter good he was vexed by defects of writing, which he described as "not mere inelegance, but such as to perplex and even travesty or hide the author's meaning." He therefore prepared a set of "Notes on the Composition of Scientific Papers," a work giving evidence of Allbutt's zeal in the education of physicians, for in it are carefully exposed the faults that writers are prone to commit. The book can serve as the memorial of a master of the most elegant English who descended to the task of teaching ordinary students how to write.

In September 1904, while on a visit to this country, he attended the meeting of the Congress of Fine Arts and Sciences, at the St. Louis

18. Allbutt, Thomas Clifford: Science and Medieval Thought, London, C. J. Clay & Sons, 1901.

19. Allbutt, Thomas Clifford: Historical Relations of Medicine and Surgery to the End of the Sixteenth Century, New York, Macmillan & Co., 1905.

20. Allbutt, Thomas Clifford: Greek Medicine in Rome, New York, The Macmillan Company, 1921.

21. Allbutt, Thomas Clifford: Byzantine Medicine, New York, The Macmillan Company, 1921.

Exposition. He was in charge of the division of internal medicine. His lecture on "Historical Relations" established among Americans a feeling of deep respect for the great scholar from England.

In October of the same year at the opening of the new clinical amphitheater at the Johns Hopkins Hospital, where William Osler, his "brother Regius," was to teach for only a few months longer, he made an address in which he defended the classics and suggested that conversation in Greek and Latin should be practiced despite the fact that they are "dead" languages. It is natural that he should have had such a view, because in his university days he had won among other valuable scientific awards a scholarship for classics.

In "Science in School," published in 1917, he incorporated his theories of training in science and suggested reforms in the system current in England.

In 1898 he visited America again to deliver the Lane Lectures at the Cooper College in San Francisco. His subject was "The Cardiovascular System." He then sailed to Honolulu and Japan. On his return to America he kept numerous engagements, including the lecture at Johns Hopkins University on medicine in the nineteenth century, and had one of the most enjoyable experiences of his life with Colonel Billings at the New York Public Library. In 1906 he made a hurried visit to Canada, where he received the degree of Doctor of Laws from the University of Toronto and from Magill University.

In 1920 he was president of the British Medical Association. At that time he was presented by his British colleagues with his portrait painted by William Orpen. Some years later a bronze portrait of him was unveiled in the library of the medical school in Downing Street, Cambridge.

His death occurred early on Sunday morning, Feb. 22, 1925, at the end of a very rich life. His body was buried at Trumpington, near Cambridge, on the following Wednesday. The funeral services were held in the chapel of Caius College, at the same time as a memorial service held in Great St. Mary's, the university church.

His estate was valued at £56,963. He bequeathed his residuary estate to his old college at Cambridge, "to establish a Fellowship for medical research or otherwise for the benefit of the College provided no part be made for buildings" and "that £50 each go to the Medical Charitable Fund for the West Riding of Yorkshire and the British Medical Benevolent Fund." To the Fitzwilliam Museum in Cambridge he left his portrait by Orpen, and on the death of Lady Allbut a quantity of antique furniture and drawings and paintings by noted artists is to pass into the possession of this museum.

In 1869 Allbutt married Susan England, the daughter of Thomas England. No children were born to them. Lady Allbutt survives Sir Clifford, and she sent to me the early photograph of her distinguished husband, which is here reproduced.

Allbutt's biographer, Sir Humphry Davy Rolleston,[22] summed up his life somewhat as follows: Allbutt's life span covered perhaps the most fascinating period of the development of medical science. He was born before the introduction of anesthesia; he saw the first advances in bacteriology and immunology and of antiseptic and aseptic surgery. He aided considerably in the establishment of nursing as a profession and was one of the first to recognize the value of public health and preventive medicine; he proposed the open-air treatment for tuberculosis and for certain fevers; he witnessed the invention of the roentgen rays and the discovery of radium. In his mature years the World War was fought. He was at once alert to the introduction of the new neurology, cardiology and biochemistry. He welcomed the advance of endocrinology. No physician during the last fifty years of his life was better remembered than was Sir Clifford Allbutt. Of none is the memory more precious to his profession.

Allbutt himself made many additions to the science and art of medicine, some of which have become so commonplace as to have lost the identity of their association with his name. He declared:

> The basis of our present professional learning might be studied with advantage not so much out of historical curiosity, but in order to show for future edification how symptoms and schools of thought arise, and how their votaries make good in some directions and how the unimportant as well as the incorrect fall gradually away from the store of knowledge leaving us what is valuable to base future developments.

It was along such broad and fundamental lines that the mind of Allbutt ever worked.

While Allbutt's death was a great loss to science, as well as to England, his life was a tremendous asset to the entire world.

> What splendid service Allbutt rendered to medical science and practice in England by precept and example, by his own ever-graceful and thoughtful writings, by the stimulus he imparted to other workers in the field. A truly great physician with healing in his presence and a courteous suavity in all he said and did. Rare is the blending of intellectual moral and aesthetical qualities that he displayed and this generation will not look upon his like again.

He possessed an unrivaled knowledge of the literary side of medicine. It has been well said of him that "from time to time medical science

22. Rolleston, Humphry Davy: The Right Honourable Sir Thomas Clifford Allbutt, K.C.B., M.A., M.D., F.R.C.P., F.R.S., Hon. M.D., D.Sc., D.C.L., LL.D., Regius Professor of Physic in the University of Cambridge: A Memoir, New York, The Macmillan Company, 1929, p. 296.

has the good fortune to possess a person who, through extensive knowledge and acute judgment and by a phenomenal gift of expression is able to link the past with the present, and to record the history of medical science thereby becoming an important agent whose efforts make the objective sciences more humane." Such a man was Sir Thomas Clifford Allbutt.

Like Jonathan Hutchinson, Allbutt was a "forward-looking" man. In his Harveian Oration, "On Science and Medical Thought," delivered in October 1900, Allbutt declared: "We celebrate the memory of great men in the certain hope that in their children they will be born again." And at another time he stated: "We are living thankfully in a glorious time; our children will go much further still but they will owe much of their progress to the principles laid down by the great men of our own times."

Since Allbutt's bent was artistic rather than scientific, it was not expected by those who observed him in his early years that he would acquire a commanding reputation among physicians of the world. A quotation from an article in the *British Medical Journal* for 1883 epitomizes his creed: "The best doctor is the best artist and the best medical artist is the master and not the servant of his science." [23]

The artistic element in his nature was never lost or even subdued but sought expression by his writings in which his artistic feeling played a part in his literary ability so manifest in his medical, scientific and historical writings, as well as in his occasional speeches in his later years.

Allbutt was a leader of the medical profession in England throughout his days; his position was unique in the medical world. A great physician, great scholar, great teacher and great writer, in his own generation he can be compared only with Sir William Osler, but in his finished literary style he surpassed the latter, as he did every one else. Sir Berkeley Moynihan paid this tribute to him at the University of Leeds (1923-1924):

> Sir Clifford Allbutt, the most deeply learned physician of this day, master of a style of English which for sheer beauty and majesty is perhaps unmatched by that of any other scientific author of our generation; an Orator whose speech makes time seem hasty; a cultured upright English gentleman is the pride of the school he served so long and so well.

Allbutt readily appreciated the work of others and was always sympathetic toward all honest work, even though he might not feel free to commend the result. He was a keen critic of any doctrine brought forward, and his criticisms when called for were boldly stated and unfalteringly expressed. He possessed a remarkably flexible mind and was always progressive in his views and writings. To the last he kept

23. Allbutt, Thomas Clifford: Brit. M. J. **2**:664, 1883.

abreast of the times and was ever ready to encourage other workers, pointing out to them where the labor might bear fruit. Up to the week before his death his mind was keenly alert on the salient points in any discussion in the contemporary medical journals, and he died in the full tide of his activities. He was to have lectured at the Royal Society in the autumn of 1925.

His frequent letters to the London *Times* served to endear him to the hearts of laymen, who found helpful advice and criticism in his contributions. In the number of the *Lancet* containing his obituary is a letter from him on "Alkalies in Certain Diseases of the Skin," evoked by a recent article in that journal. To the end he declared that "only two things are essential—to live uprightly and to be wisely industrious." The guiding star of his life, he used to say, was industry, and he declared that any little success that he might have had was due to constant application; yet, he thoroughly believed that a physician should take a continuous holiday of six weeks; he once missed doing so and regretted it ever after. He had traveled extensively, visiting Greece and Rome and other Italian cities along the Mediterranean, as well as Bayreuth and the Scandinavian countries.

For many years—indeed, from his fourteenth year and until he was in his ninetieth year—he had been famous as an alpinist and was well known in the north of England, especially in the lake region, for his long walks through that rough country. Osler, in 1918, recorded that he found Sir Clifford in fine form and that despite his 82 years he was exhibiting extraordinary physical power, thinking nothing of cycling 15 miles (24.2 kilometers) daily! Except for deafness, age seemed not to affect his faculties, while his subtle intellect was keen to the end of his long life.

Clifford Allbutt was distinguished in looks as well as in activities. He had a wonderful ability to deal with men and things, and every one who came in contact with him was deeply impressed by his keen intelligence, culture and urbanity. He was often referred to as the "spiritual aristocrat of medicine." His courtesy was unfailing to all, high or low, and, like other qualities, was a finished product. In his personal relations he was one of the kindliest of men, and despite the exalted position to which he was placed by acclamation of his fellows he was to his last hours without a trace of that arrogance which success engenders in lesser minds. In spite of all this he must have realized that he had become one of the most honored and beloved of the heads of the medical profession and had largely contributed to the advancement of medical science. Like the true Yorkshireman that he was, he was conscious of his own worth, yet so gracious and urbane was he that one would never have suspected him of being one of that "peculiar" county, although under the polish the grit was there.

He was a much desired conversationalist and lecturer. His home was the center of scientific and literary exchange, as he was well acquainted with many famous authors, both general and scientific. He was on intimate friendly terms with the Brontë sisters, and he was supposed to have inspired the character Tertius Lydgate in George Eliot's "Middlemarch." Osler,[24] in commenting on this, declared that although George Eliot had frequent occasions for contact with Sir Clifford, there was nothing in his career in common with that of the famous character except their training and their high aspirations. It is likely that Allbutt's early course at Leeds had given the gifted authoress a suggestion for the portrayal of a character in her novel.

When one contemplates the activities of this long life one is struck by Allbutt's deep and all-absorbing interest in ophthalmology and his giving out to the world for many years the product of his study. It was an episode in his life—a mere incidental reaction to the call for an understanding of great medical problems. That he did not continue with the special practice of ophthalmology when the time in his affairs seemed ripe for him to confine himself to that branch is an unsolved mystery. That he should have accomplished what he did exemplifies the well known characteristics of Yorkshire perseverance and strenuous devotion to whatever work one has at hand. Allbutt was quick to seize the opportunity offered by Dr. Ogle's suggestion that the ophthalmoscope might be of service in the diagnosis of intracranial disorders. Allbutt was never afraid of work, and despite the unwillingness of many practitioners to assist and their timidity to persist in studying the eyes he was never doubtful of success and never weary in carrying out whatever task he undertook.

In his interest in ophthalmology Allbutt might be likened to one of the early navigators who ventured out in voyaging to an unexplored land. There was a "point of departure," but no "course and distance" could be defined prior to his sailing. A "landing" was to be made somewhere, yet because his instruments were imperfect, much of navigation was accomplished by "plane sailing." But, like the captain, Allbutt, being a careful navigator, made frequent "observations" to ascertain his position; the results he "plotted" from time to time before the voyage was over; a "track" was lined off on the "chart" and the details recorded in his "log book." A true course having been laid, it was possible for him, as for the captain, to return to the point of departure, and a good account was given of the voyage, with a descrip-

24. Osler, cited in Cushing, Harvey: Life of Sir William Osler, Oxford, Clarendon Press, 1925, vol. 1, p. 463.

tion of the newly explored country. Allbutt's ophthalmoscope was his "observation-instrument"; his discussions and writings became his chart and his log book.

So satisfied was this "navigator" with the product of his voyage and so willing was he that others should share in his rewards that he widely opened the way for any who wished to make the same explorations. Although he himself never set out again for his early field, what he learned by his sailing he faithfully applied in all his later voyaging.

Present day ophthalmologists, Allbutt's successors and followers, are the later voyagers into that continent so freely opened out to them. His unwillingness to pursue ophthalmology as a source of special medical practice is one of the remarkable phenomena in the history of modern medicine. I have undertaken this memoir lest the part he played in this drama might become forgotten by ophthalmologists in this generation and the next.

The materials for this memoir were gathered from many sources and accumulated over a number of years. In the removal of my library from one house to another, in March, the list of references was mislaid and has not been recovered. Each of Allbutt's published papers and reports and the comments and discussions offered by contemporaries were read and analyzed; the epitome was included in what appears here. I failed to find Allbutt's name in the index of Hirschberg's "Geschichte der Augenheilkunde" [25] and in Wilbrand and Saenger's "Die Neurologie des Auges." [26]

I had never met Sir Clifford and had only a brief correspondence with him. I therefore have had to rely on numerous personal letters received from Sir James Crichton-Browne and others who had known him. Much was taken from Sir Humphry Davy Rolleston's "Memoir."

The object of this series of "Short Studies on the History of Ophthalmology" has been to tell of the introduction of ophthalmoscopy into medical practice and to present memorials of those who did so much through ophthalmoscopic investigations in the creation of modern medicine. So far as concerned England, the earliest days of the ophthalmoscope have been recounted and the lives of Hutchinson, Hughlings Jackson and Clifford Allbutt presented. The status of their contribution

25. Hirschberg, J.: Geschichte der Augenheilkunde, in von Graefe, T., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, Leipzig, W. Engelmann, 1915.

26. Wilbrand, H., and Saenger, A.: Die Neurologie des Auges, Wiesbaden, J. F. Bergmann, 1900.

might be expressed by the following testimony from Dr. W. H. Broadbent,[27] Physician to St. Mary's Hospital, London:

> The ophthalmoscope, thanks to the efforts of Dr. Clifford Allbutt and Dr. Hughlings Jackson, may be said fairly to have taken its place in English medicine; and it is scarcely possible to over state the additional interest it has brought into the study of the diseases of the nervous system. It has removed causes of confusion and error; it has opened absolutely new sources of information as to the state of the intracranial circulation, which will eventually conduce greatly to decision in treatment. It has, also, or rather the far-reaching vision of the men who have forced it upon the attention of the profession, widened the horizon of pathological research by stating new questions of various kinds for solution.

317 South Fifteenth Street.

27. Broadbent, W. H.: On the Causation and Significance of the Choked Disk in Intracranial Diseases, Brit. M. J. **1**:633, 1872.

EFFECT OF FATIGUE ON THE ADJUSTMENT OF THE EYE TO NEAR AND FAR VISION

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In previous work done with the tachistoscope in the measurement of the speed of adjustment of the eye to near and far vision I [1] reported only the normal findings. Since that time I have been able to accumulate data for tests of the dynamic speed of vision for pilots with fatigue after one, two, three and four hours of flying.[2]

I shall first show the results of six hundred and twenty-eight tests on aviators under normal conditions in the three phases of speed of accommodation, i. e., the speed of accommodation from near to far, the speed of accommodation from far to near, and the speed of accommodation from near to far and return to near.

This is to demonstrate what I consider the normal limits for subjects arranged according to age in five groups and to show the causes for the data which are not within those limits.

In all the tables in this paper the pilots are arranged in five year groups progressing from 20 to 50 years of age, as noted in the left column of the tables. The time is given in fractions of seconds, in progressions of one-tenth second from left to right at the top of tables 1 to 8. The average time for each five year group and the number of tests for each five year group are noted in the last two right columns of tables 1 to 8. The total number of tests carried out in each group of 0.1 second is noted at the bottom of the tables.

The total number of tests and the average time for all the age groups are noted in the right lower corner of tables 1 to 8.

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* In care of Postmaster.

1. Robertson, C. J.: (*a*) Measurement of the Speed of Adjustment of the Eye to Near and Far Vision, U. S. Nav. M. Bull. **32**:275-283 (July) 1934; (*b*) A Comparative Study of the Measurement of the Speed of Adjustment of the Eye for Near and Far Vision, ibid. **33**:187-205 (April) 1935; (*c*) Measurement of Speed of Adjustment of Eye to Near and Far Vision, Arch. Ophth. **14**:82-89 (July) 1935; (*d*) Measurement of Speed of Adjustment of Eye to Near and Far Vision: A Further Study, ibid. **15**:423-434 (March) 1936.

2. (*a*) Ferree, C. E., and Rand, G.: An Instrument for Measuring Dynamic Speed of Vision, Speed of Accommodation, and Ocular Fatigue, Arch. Ophth. **15**: 1072-1687 (June) 1936. (*b*) Aviation Physical Examination, United States Navy Standard, 1927, in the Manual of the Medical Department, United States Navy, Washington, D. C., Government Printing Office, 1927.

In the table showing the speed of accommodation from near to far the heavy line beginning at 0.9 second in the age group of persons from 20 to 24 years old and increasing 0.1 second with each five year group is the demarcation between what is considered the normal and the abnormal. It is considered that all the tests listed to the right of this line showed pathologic conditions. In the table showing the speed of accommodation from far to near, the heavy line begins at 0.6 second, and in that showing the speed of accommodation from near to far and return to near, at 1.35 seconds.

Table 1 shows the speed of accommodation from near to far. The maximum number of tests for all the age groups is found to be in the time range from 0.7 to 0.79 second. The progressive increase of

TABLE 1.—*Speed of Accommodation of Pilots from Near to Far*

	Time, Seconds											
Age, Years	0.30 to 0.39	0.40 to 0.49	0.50 to 0.59	0.60 to 0.69	0.70 to 0.79	0.80 to 0.89	0.90 to 0.99	1.0 to 1.09	1.1 to 1.19	1.2 On	Number of Tests	Average Time for Age Group, Seconds
20 to 24	..	..	6	33	33	14	4	..	..	..	90	0.713
25 to 29	..	..	19	96	125	45	14	4	2	..	305	0.739
30 to 34	..	..	2	36	55	40	15	6	3	..	157	0.783
35 to 39	..	..	1	6	19	15	10	3	2	1	57	0.817
40 to 44	..	..	..	1	3	3	3	3	1	..	14	0.897
45 to 49	..	..	..	..	..	2	..	..	2	1	5	1.135
Total	..	..	28	172	235	119	46	16	16	2	628	
Average time for all age groups, seconds												0.846

time, as well as the average time, is demonstrated for each five year age group. The average time for all the age groups is 0.846 second.

The tests listed at the right of the line are fourteen in number and are considered to have shown pathologic conditions. The number of these tests in each age group and the types of conditions are taken up. Each subject may have one or more of the conditions noted.

In the group of subjects from 20 to 24 years old, inclusive, the tests for determining the speed of accommodation from near to far in the time range beyond 0.9 second showed pathologic conditions in four cases. These conditions were: inequality in accommodation, right and left hyperphoria near the outside limits, exophoria of 12 diopters or more at 33 cm., inequality of visual acuity, visual acuity of less than 20/20 in each eye and astigmatic error.

In the group of subjects from 25 to 29 years old, inclusive, the tests for the determination in the time range beyond 1 second showed pathologic conditions in six cases. The conditions were: exophoria of

8 diopters or more at 33 cm., astigmatic error, myopia, inequality of visual acuity, an angle of convergence near the lower limits of normal and visual acuity of less than 20/20 in each eye.

In the group of subjects from 30 to 34 years old, inclusive, the tests for the determination in the time range beyond 1.1 seconds showed pathologic conditions in three cases. The conditions were: astigmatic error, exophoria of 8 diopters or more at 33 cm. and visual acuity of less than 20/20.

In the group of subjects from 35 to 39 years old, inclusive, the tests carried out in the time range beyond 1.2 seconds showed pathologic conditions in one case. The conditions were: visual acuity of less than 20/20 and exophoria of 10 diopters or more at 33 cm.

TABLE 2.—*Speed of Accommodation of Pilots from Far to Near*

	Time, Seconds											
Age, Years	0.30 to 0.39	0.40 to 0.49	0.50 to 0.59	0.60 to 0.69	0.70 to 0.79	0.80 to 0.89	0.90 to 0.99	1.0 to 1.09	1.1 to 1.19	1.2 On	Number of Tests	Average Time for Age Group, Seconds
20 to 24	..	55	29	6	..	..	..	..	..	..	90	0.475
25 to 29	9	158	106	32	..	..	..	..	..	..	305	0.503
30 to 39	1	45	63	40	6	1	1	..	..	..	157	0.538
35 to 39	..	12	21	10	9	3	1	1	..	..	57	0.604
40 to 44	..	..	..	6	3	4	..	..	1	..	14	0.771
45 to 49	..	..	..	..	1	2	..	..	..	2	5	1.188
Total	10	270	219	94	19	10	2	1	1	2	628	
Average time for all age groups, seconds												0.663

Table 2 shows the speed of accommodation from far to near. The maximum number of tests for all the age groups is found in the time range from 0.4 to 0.49 second, as compared to the time range of 0.7 to 0.79 second in the phase of speed of accommodation from near to far. The progressive increase of time, as well as the average time, is shown for each five year age group. The average time for all the age groups is 0.663 second, as compared to 0.846 second in the phase of accommodation from near to far.

The tests listed at the right of the heavy line are thirteen in number and are considered to have shown pathologic conditions. The number of these tests in each age group and the types of conditions are taken up. Each subject may have one or more of the conditions noted.

In the group of subjects from 20 to 24 years old, inclusive, the tests for determining the speed of accommodation from far to near in the time range beyond 0.6 second showed pathologic conditions in

six cases. The pathologic conditions were: inequality of visual acuity, inequality of accommodation, astigmatic error and exophoria of 10 diopters at 33 cm.

In the group of subjects from 25 to 29 years old, inclusive, no test carried out in the time range beyond 0.7 second showed pathologic conditions.

In the group of subjects from 30 to 34 years old, inclusive, the tests for the determination in the time range beyond 0.8 second showed exophoria of 10 diopters at 33 cm. in two cases.

In the group of subjects from 35 to 39 years old, inclusive, the tests for the determination in the time range beyond 0.9 second showed visual acuity of less than 20/20 in two cases.

TABLE 3.—*Speed of Accommodation of Pilots from Near to Far and Return to Near*

	Time, Seconds											
Age, Years	0.95 to 1.049	1.05 to 1.149	1.15 to 1.249	1.25 to 1.349	1.35 to 1.449	1.45 to 1.549	1.55 to 1.649	1.65 to 1.749	1.75 to 1.849	1.85 On	Number of Tests	Average Time for Group, Seconds
20 to 24	7	17	33	24	9	..	..	..	..	..	90	1.171
25 to 29	21	40	102	95	36	9	2	..	..	..	305	1.239
30 to 34	1	9	27	48	35	26	5	4	1	1	157	1.329
35 to 39	..	2	7	11	12	9	8	6	1	1	57	1.421
40 to 44	..	..	..	1	1	2	2	4	1	3	14	1.668
45 to 49	..	..	..	..	..	..	..	1	1	3	5	2.323
Total	29	68	169	179	93	46	17	15	4	8	628	
Average time for all age groups, seconds												1.526

In the group of subjects from 40 to 44 years old, inclusive, the tests for the determination in the time range beyond 1 second showed pathologic conditions in one case. These were exophoria of 10 diopters at 33 cm. and inequality of accommodation.

In the group of subjects from 45 to 49 years old, inclusive, the tests for the determination in the time range beyond 1.1 seconds showed pathologic conditions in two cases. The conditions were: astigmatic error in one eye, astigmatism in each eye and exophoria of 10 diopters at 33 cm.

Table 3 shows the speed of accommodation from near to far and return to near. The maximum number of tests is found to be in the time range of from 1.25 to 1.349 seconds. The progressive increase of time, as well as the average time, is demonstrated for each five year group. The average time for all the age groups is 1.526 seconds.

The tests listed at the right of the heavy line are forty-six in number and are considered to have shown pathologic conditions. The

number of these cases in each age group and the types of conditions are taken up. Each subject may have one or more of the conditions noted.

In the group of subjects from 20 to 24 years old, inclusive, the tests for determining the speed of accommodation from near to far and return to near in the time range beyond 1.35 seconds showed pathologic conditions in nine cases. The conditions were: exophoria of from 8 to 12 diopters at 33 cm., left hyperphoria near the extreme limit, inequality of visual acuity, unequal accommodation, astigmatism, visual acuity of less than 20/20 and myopia.

In the group of subjects from 25 to 29 years old, inclusive, the tests for the determination in the time range beyond 1.45 seconds showed pathologic conditions in eleven cases. The conditions were: exophoria of 10 diopters or more at 33 cm., astigmatic error, inequality of accommodation, myopia, visual acuity of less than 20/20 and inequality of visual acuity.

In the group of subjects from 30 to 34 years old, inclusive, the tests for the determination in the time range beyond 1.55 seconds showed pathologic conditions in eleven cases. The conditions were: exophoria of from 8 to 10 diopters at a distance of 33 cm., exophoria of 10 diopters or more at 33 cm., astigmatism, inequality of accommodation and visual acuity of less than 20/20.

In the group of subjects from 35 to 39 years old, inclusive, the tests for the determination in the time range beyond 1.65 seconds showed pathologic conditions in eight cases. The conditions were: inequality of visual acuity, astigmatism and visual acuity of less than 20/20 in one or both eyes.

In the group of subjects from 40 to 44 years old, inclusive, the tests for the determination in the time range beyond 1.75 seconds showed pathologic conditions in four cases. The conditions were: exophoria of 10 diopters at 33 cm., astigmatic error and exophoria of 12 diopters at 33 cm.

In the group of subjects from 45 to 49 years old, inclusive, the tests for the determination in the time range beyond 1.85 seconds showed pathologic conditions in three cases. The conditions were: visual acuity of less than 20/20 cm. and exophoria of 14 diopters at 33 cm.

In a brief résumé of the cases in which the speed of accommodation to near and far vision was considered below the limits of safety the cause for the loss in speed is deemed evident and proved. It is considered that as age progresses the pilots with known ocular conditions are less and less able to overcome the conditions and thus become an aeronautic menace. Certain conditions can be improved with proper treatment.

It is noted that many pilots have a pathologic condition of the eyes but do not show a loss in speed of accommodation. These pilots are in the younger age groups, and it is considered that by virtue of their youth they are able to overcome the condition, but they should be carefully watched as age progresses.

Of the visual requirements in aviation as laid down in the Manual of the Medical Department of the United States Navy,[2b] the following conditions did not show in my tests as pathologic: esophoria and exophoria at a distance of 6 meters, prism divergence and depth perception.

It is considered that the following conditions definitely cause a slowing of the speed of adjustment of the eye to near and far vision: age; exophoria of 8 diopters or more at 33 cm.; astigmatism of one or both eyes; visual acuity below 20/20 in one or both eyes; inequality of visual acuity, even though vision is above the required 20/20; myopia, an angle of convergence near the lower limits of normal and a sluggish mental reaction.

In table 4 is shown the speed of accommodation from near to far and return to near of pilots after one hour of flying compared with their normal speed of accommodation. It is not considered that one hour of flying causes severe fatigue; nevertheless it is demonstrated that even with the minor degree of fatigue resulting there is a slowing of the speed of adjustment of the eye to near and far vision. The effect of fatigue is demonstrated throughout the table, with particular reference to the average time for the age groups.

The cases in which the speed of accommodation is considered below the limit of safety are discussed separately, and the suspected cause for the slowing is given.

In the group of subjects from 20 to 24 years old, inclusive, tests for determining the speed of accommodation from near to far and return to near showed a speed below the limits of safety in four cases.

CASE 1.—No pathologic condition was demonstrable. The tachistoscopic findings were: normal speed, 1.352 seconds, and speed in the fatigued state, 1.452 seconds.

CASE 2.—No pathologic condition was demonstrable. The tachistoscopic findings were: normal speed, 1.287 seconds, and speed in the fatigued state, 1.353 seconds.

CASE 3.—Astigmatic error, visual acuity of less than 20/20, left hyperphoria of 0.75 diopter, and exophoria of 12 diopters at 33 cm. were present. The tachistoscopic findings were: normal speed, 1.386 seconds, and speed in the fatigued state, 1.562 seconds.

CASE 4.—Astigmatic error and inequality of visual acuity were demonstrated. The tachistoscopic findings were: normal speed, 1.32 seconds, and speed in the fatigued state, 1.352 seconds.

In the group of subjects from 25 to 29 years old, inclusive, tests for the determination showed speed below the limits of safety in five cases.

TABLE 4.—*Speed of Accommodation from Near to Far and Return to Near of Pilots with Fatigue After One*

TABLE 4.—*Speed of Accommodation from Near to Far and Return to Near of Pilots with Fatigue After One Hour of Flying Compared with Their Normal Speed*

Age, Years	Time, Seconds: 0.95 to 1.049		1.05 to 1.149		1.15 to 1.249		1.25 to 1.349		1.35 to 1.449		1.45 to 1.549		1.55 to 1.649		1.65 to 1.749		1.75 On		Number of Tests	Average Time for Age Group, Seconds	
	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State		Normal State	Fatigued State
20 to 24	1	1	6	5	9	9	12	12	3	2	..	1	..	1	..	..	..	..	31	1.231	1.284
25 to 29	3	1	11	7	34	21	23	32	7	15	3	5	..	..	..	..	..	..	81	1.244	1.287
30 to 34	..	1	2	1	9	2	6	12	11	10	..	4	2	..	..	..	..	..	30	1.296	1,303
35 to 39	..	..	..	..	1	1	3	1	2	1	..	3	3	2	1	..	..	2	10	1.443	1.507
40 to 44	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	1	..	1	1.925	1.628
45 to 49	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	1	1.782	2.464
Normal	4		19		53		44		23		3		5		1		2		154		
Fatigued		3		13		33		57		28		13		4		..		3			
Average time for all age groups, seconds																				1.492	1.578

TABLE 5.—*Speed of Accommodation from Near to Far and Return to Near of Pilots with Fatigue After Two Hours of Flying Compared with Their Normal Speed*

Age, Years	Time, Seconds: 0.95 to 1.049		1.05 to 1.149		1.15 to 1.249		1.25 to 1.349		1.35 to 1.449		1.45 to 1.549		1.55 to 1.649		1.65 to 1.749		1.75 On		Number of Tests	Average Time for Age Group, Seconds	
	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State		Normal State	Fatigued State
20 to 24	3	..	1	4	1	1	3	2	..	1	..	..	..	..	..	..	..	..	8	1.150	1.197
25 to 29	2	1	6	2	17	16	20	21	5	6	2	4	..	1	..	1	..	..	52	1.251	1.289
30 to 34	1	..	2	..	6	3	9	8	9	10	2	6	..	2	1	..	..	1	30	1.271	1.339
35 to 39	..	..	1	..	6	..	..	6	..	1	2	2	1	..	..	1	..	..	10	1.441	1.585
40 to 44	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	1	1	1.595	1.892
45 to 49	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.....	.....
Normal	6		10		30		32		14		6		2		1		..		101		
Fatigued		1		6		20		37		18		12		3		2		2			
Average time for all age groups, seconds																				1.341	1.459

CASE 1.—Visual acuity was 20/20 with effort. The tachistoscopic findings were: normal speed, 1.43 seconds, and speed in the fatigued state, 1.485 seconds.

CASE 2.—No pathologic condition was demonstrable. The tachistoscopic findings were: normal speed, 1.21 seconds, and speed in the fatigued state, 1.485 seconds.

CASE 3.—Exophoria of 10 diopters at 33 cm. was present. The tachistoscopic findings were: normal speed, 1.287 seconds, and speed in the fatigued state, 1.507 seconds.

CASE 4.—Inequality of accommodation and exophoria near the outside limits at a distance of 33 cm. were demonstrable. The tachistoscopic findings were: normal speed, 1.453 seconds, and speed in the fatigued state, 1.496 seconds.

CASE 5.—No pathologic condition was demonstrable. The tachistoscopic findings were: normal speed, 1.468 seconds, and speed in the fatigued state, 1.452 seconds.

In the group of subjects from 35 to 39 years old, inclusive, tests for the determination showed speed below the limits of safety in two cases.

CASE 1.—Right hyperphoria of 0.75 diopters was present. The tachistoscopic findings were: normal speed, 1.672 seconds, and speed in the fatigued state, 1.76 seconds.

CASE 2.—There was no record of findings due to age. The tachistoscopic findings were: normal speed, 1.672 seconds, and speed in the fatigued state, 1.804 seconds.

In the group of subjects from 45 to 49 years old, inclusive, the tests for the determination showed speed below the limits of safety in 1 case.

CASE 1.—No pathologic condition was demonstrable. The tachistoscopic findings were: normal speed, 1.782 seconds, and speed in the fatigued state, 2.464 seconds.

Chart 1 shows the speed of accommodation from near to far and return to near of pilots with fatigue after one hour of flying compared with their normal speed of accommodation in terms of the average time for the five year age groups. Only one test was carried out in the group of subjects from 40 to 44 years old, inclusive, and only one in that of subjects from 45 to 49 years old, inclusive. Even with this minor degree of fatigue (after one hour of flying) the slowing of speed is well noted.

Table 5 demonstrates the speed of accommodation from near to far and return to near of pilots with fatigue after two hours of flying compared with their normal speed of accommodation. It is here more evident that fatigue has a bearing on the speed of adjustment for near and far vision.

In the group of subjects from 20 to 24 years old, inclusive, the tests for determining the speed of accommodation from near to far and return to near showed a speed below the limits of safety in one case.

CASE 1.—Inequality of visual acuity and hyperopia were present. The tachistoscopic findings were: normal speed, 1.309 seconds, and speed in the fatigued state, 1.441 seconds.

In the group of subjects from 25 to 29 years old, inclusive, the tests for the determination showed a speed below the limits of safety in six cases.

CASE 1.—Myopia was present. The tachistoscopic findings were: normal speed, 1.43 seconds, and speed in the fatigued state, 1.54 seconds.

CASE 2.—Inequality of accommodation and exophoria of 10 diopters at 33 cm. were present. The tachistoscopic findings were: normal speed, 1.309 seconds, and speed in the fatigued state, 1.45 seconds.

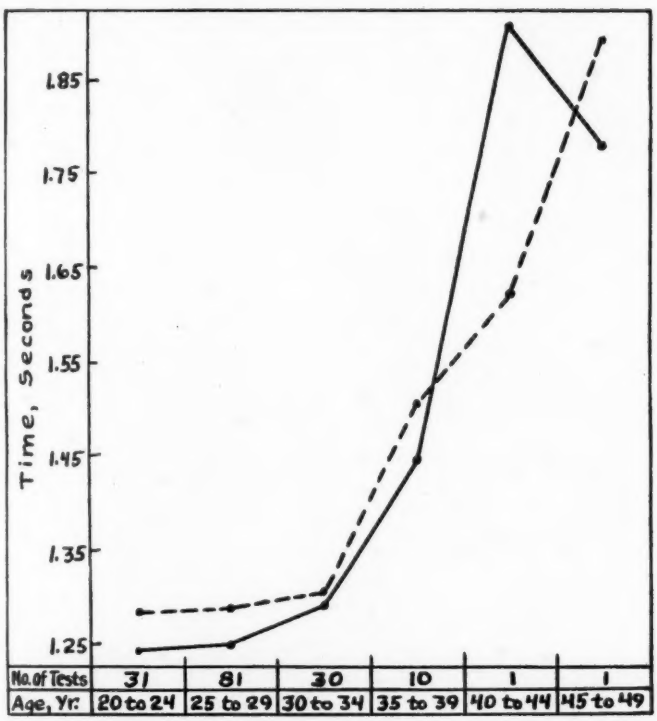

Chart 1.—Speed of accommodation from near to far and return to near of pilots with fatigue after one hour of flying (broken line) compared with their normal speed of accommodation (unbroken line), in terms of the average time for the five year age groups.

CASE 3.—Exophoria of 10 diopters at 33 cm. was present. The tachistoscopic findings were: normal speed, 1.287 seconds, and speed in the fatigued state, 1.507 seconds.

CASE 4.—No pathologic condition was demonstrable. The tachistoscopic findings were: normal speed, 1.353 seconds, and speed in the fatigued state, 1.551 seconds.

CASE 5.—Myopia was found. The tachistoscopic findings were: normal speed, 1.408 seconds, and speed in the fatigued state, 1.606 seconds.

Case 6.—No pathologic condition was demonstrable. The tachistoscopic findings were: normal speed, 1.496 seconds, and speed in the fatigued state, 1.496 seconds.

In the group of subjects from 30 to 34 years old, inclusive, the tests for the determination showed a speed below the limits of safety in three cases.

Case 1.—No pathologic condition was demonstrable. The tachistoscopic findings were: normal speed, 1,364 seconds, and speed in the fatigued state, 2,277 seconds.

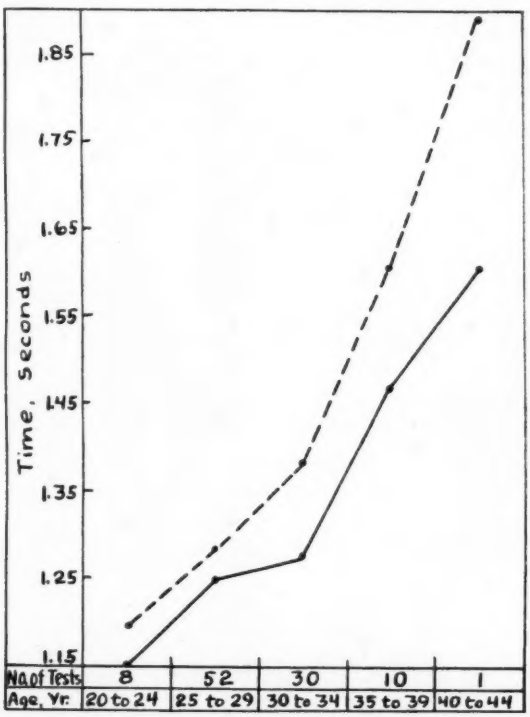

Chart 2.—Speed of accommodation from near to far and return to near of pilots with fatigue after two hours of flying (broken line) compared with their normal speed of accommodation (unbroken line), in terms of the average time for the five year age groups.

Case 2.—No pathologic condition was demonstrable. The tachistoscopic finding were: normal speed, 1.716 seconds, and speed in the fatigued state, 1.87 seconds.

Case 3.—Inequality of visual acuity and exophoria of 9 diopters at 33 cm. were present. The tachistoscopic findings were: normal speed, 1.43 seconds, and speed in the fatigued state, 1.606 seconds.

In the group of subjects from 35 to 39 years old, inclusive, the test for the determination showed a speed below the limits of safety in one case.

TABLE 6.—*Speed of Accommodation from Near to Far and Return to Near of Pilots with Fatigue After Three Hours of Flying Compared with Their Normal Speed*

Age, Years	Time, Seconds: 0.95 to 1.049		1.05 to 1.149		1.15 to 1.249		1.25 to 1.349		1.35 to 1.449		1.45 to 1.549		1.55 to 1.649		1.65 to 1.749		1.75 On		Number of Tests	Average Time for Age Group, Seconds	
	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State		Normal State	Fatigued State
20 to 24	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.....	.....
25 to 29	..	..	3	1	..	1	1	2	..	..	..	..	..	..	..	..	..	..	4	1.121	1.231
30 to 34	..	..	1	1	4	1	6	6	1	2	2	2	1	3	..	..	..	..	15	1.321	1.380
35 to 39	..	..	..	..	1	1	1	..	1	1	1	1	..	1	..	..	1	1	5	1.470	1.575
40 to 44	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.....	.....
45 to 49	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.....	.....
Normal	..		4		5		8		2		3		1		..		1		24		
Fatigued		..		2		3		8		3		3		4		..		1			
Average time for all age groups, seconds																				1.304	1,396

TABLE 7.—*Speed of Accommodation from Near to Far and Return to Near of Pilots with Fatigue After Four Hours of Flying Compared with Their Normal Speed*

Age, Years	Time, Seconds: 0.95 to 1.049		1.05 to 1.149		1.15 to 1.249		1.25 to 1.349		1.35 to 1.449		1.45 to 1.549		1.55 to 1.649		1.65 to 1.749		1.75 On		Number of Tests	Average Time for Age Group, Seconds	
	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State		Normal State	Fatigued State
20 to 24	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.....	.....
25 to 29	..	..	..	2	2	1	2	1	..	..	..	..	..	..	..	..	..	..	4	1.234	1.242
30 to 34	..	..	1	..	2	1	1	3	1	1	..	..	..	1	1	..	..	..	6	1.286	1.361
35 to 39	..	..	..	..	..	..	1	..	..	1	1	1	..	..	..	..	..	..	2	1.424	1.485
40 to 44	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	1	..	..	1	1.542	1.683
45 to 49	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.....	.....
Normal	..		1		4		4		1		2		..		1		..		13		
Fatigued		..		2		2		4		2		1		1		1		..			
Average time for all age groups, seconds																				1.370	1.442

Inequality of visual acuity and vision of less than 20/20 were found. The tachistoscopic findings were: normal speed, 1.595 seconds, and speed in the fatigued state, 1.716 seconds.

In the group of subjects from 40 to 44 years old, inclusive, the tests for the determination showed a speed below the limits of safety in one case.

Inequality of accommodation and exophoria of 9 diopters at 33 cm. were present. The tachistoscopic findings were: normal speed, 1.595 seconds, and speed in the fatigued state, 1.892 seconds.

Chart 2 shows the speed of accommodation from near to far and return to near of pilots with fatigue after two hours of flying compared with their normal speed of accommodation, in terms of the average time

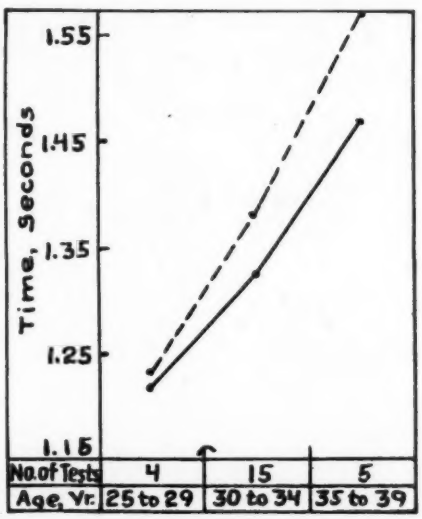

Chart 3.—Speed of accommodation from near to far and return to near of pilots with fatigue after three hours of flying (broken line) compared with their normal speed of accommodation (unbroken line), in terms of the average time for the five year age groups.

for each five year age group. This demonstrates the more noted slowing of the speed with greater fatigue, particularly in the later age groups.

Table 6 demonstrates the speed of accommodation from near to far and return to near of pilots with fatigue after three hours of flying compared with their normal speed of accommodation. Again the effect of fatigue is shown.

In the group of subjects from 30 to 34 years old, inclusive, the tests for the determination showed a speed below the limits of safety in three cases.

Case 1.—No pathologic condition was demonstrable. The tachistoscopic findings were: normal speed, 1.496 seconds, and speed in the fatigued state, 1.617 seconds.

TABLE 8.—*Speed of Accommodation from Near and Far and Return to Near of Pilots with Fatigue After from One to Four Hours of Flying Compared with Their Normal Speed*

	Time, Seconds																			
	0.95 to 1.049		1.05 to 1.149		1.15 to 1.249		1.25 to 1.349		1.35 to 1.449		1.45 to 1.549		1.55 to 1.649		1.65 to 1.749		1.75 On		Number of Tests	Average Time for Age Group, Seconds
Age, Years	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State	Normal State	Fatigued State		Normal State	Fatigued State
20 to 24	4	1	7	9	10	10	15	14	3	3	..	1	..	1	..	..	..	..	39	1.190	1.240
25 to 29	5	1	20	12	53	41	46	53	12	23	5	9	..	2	..	..	..	..	141	1.212	1.274
30 to 34	1	1	6	1	21	7	22	29	22	24	4	11	3	7	2	..	..	1	81	1.293	1.345
35 to 39	..	..	1	..	7	2	6	6	3	4	4	8	4	2	1	2	1	3	37	1.445	1.538
40 to 44	..	..	..	..	..	..	..	..	..	..	1	..	1	1	..	1	1	1	3	1.686	1.701
45 to 49	..	..	..	.	..	..	..	..	..	..	..	..	..	..	..	..	1	1	1	1.781	2.464
Normal	10		34		91		89		40		14		8		3		3		292		
Fatigued		3		22		60		102		54		29		13		3		6			
Average time for all age groups, seconds																				1.408	1.456

TABLE 9.—*Speed of Accommodation from Near to Far of Pilots and Nonpilots and of Pilots with Fatigue After One, Two, Three and Four Hours of Flying and Under Normal Conditions, in Terms of the Average for Each Age Group*

Age, Years	Speed of Pilots, Sec.	Number of Tests	Speed of Nonpilots, Sec.	Number of Tests	Speed After 1 Hr. of Flying, Sec.	Number of Tests	Normal Speed	Speed After 2 Hrs. of Flying, Sec.	Number of Tests	Normal Speed	Speed After 3 Hrs. of Flying, Sec.	Number of Tests	Normal Speed	Speed After 4 Hrs. of Flying, Sec.	Number of Tests	Normal Speed	Speed After from 1 to 4 Hrs. of Flying, Sec.	Number of Tests	Normal Speed	Total Number of Tests for Age Group
20 to 24	0.713	90	0.720	53	0.726	31	0.760	0.704	8	0.665	.....	..	.....	.....	..	.....	0.715	39	0.712	221
25 to 29	0.739	305	0.791	21	0.736	81	0.732	0.747	52	0.747	0.706	4	0.657	0.706	4	0.750	0.723	141	0.721	608
30 to 34	0.783	157	0.802	22	0.776	30	0.777	0.775	30	0.735	0.774	15	0.759	0.771	6	0.777	0.774	81	0.762	341
35 to 39	0.817	57	0.811	5	0.807	10	0.816	0.873	5	0.816	0.926	5	0.897	0.825	2	0.830	0.857	27	0.839	116
40 to 44	0.897	19	0.967	10	0.902	1	1.045	0.924	1	0.825	.....	..	.....	0.737	1	0.770	0.859	3	0.880	30
45 to 49	1.135	5	1.012	1	1.254	1	0.902	.....	..	.....	.....	..	.....	.....	..	.....	1.254	1	0.902	8
Total.....		628		112		159			101			24			13			292		1,324
Total aver.	0.844		0.850		0.866		0.838	0.804		0.757	0.802		0.770	0.759		0.781	0.807		0.782	

CASE 2.—Astigmatic error, inequality of visual acuity and vision of less than 20/20 in one eye were present. The tachistoscopic findings were: normal speed, 1.573 seconds, and speed in the fatigued state, 1,551 seconds.

CASE 3.—Inequality of accommodation and exophoria of 9 diopters at 33 cm. were found. The tachistoscopic findings were: normal speed, 1.43 seconds, and speed in the fatigued state, 1.617 seconds.

In the group of subjects from 35 to 39 years old, inclusive, the tests for the determination showed a speed below the limits of safety in one case.

CASE 1.—Visual acuity of less than 20/20, inequality of visual acuity and exophoria of 10 diopters at 33 cm. were present. The tachistoscopic findings were: normal speed, 2.2 seconds, and speed in the fatigued state, 2.002 seconds.

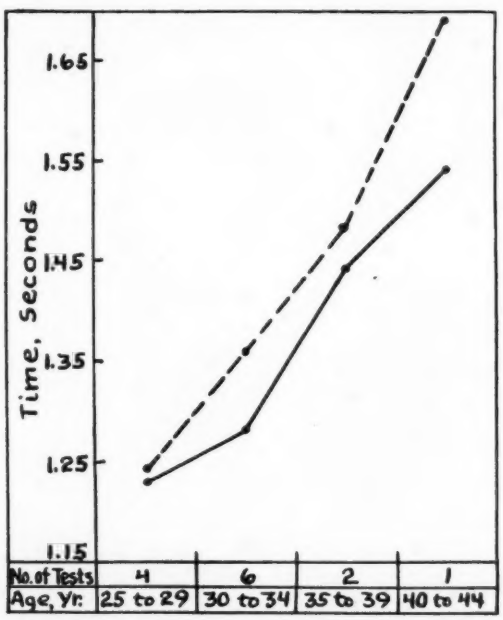

Chart 4.—Speed of accommodation from near to far and return to near of pilots with fatigue after four hours of flying (broken line) compared with their normal speed of accommodation (unbroken line), in terms of the average time for the five year age groups.

Chart 3 demonstrates the speed of accommodation from near to far and return to near of pilots with fatigue after three hours of flying with their normal speed, in terms of the average time for the five year age groups.

Table 7 shows the speed of accommodation from near to far and return to near of pilots with fatigue after four hours of flying compared with their normal speed of accommodation. As there were only thirteen tests in this study it is difficult to draw conclusions of moment. However, the effect of fatigue is definitely demonstrated.

Chart 4 shows the speed of accommodation from near to far and return to near of pilots with fatigue after four hours of flying compared with the normal speed of accommodation of the same pilots, in terms of the average time for the five year age groups.

Chart 5 shows the speed of accommodation from near to far and return to near of pilots with fatigue after from one to four hours of flying compared with their normal speed of accommodation, in terms of the average time for the five year age groups.

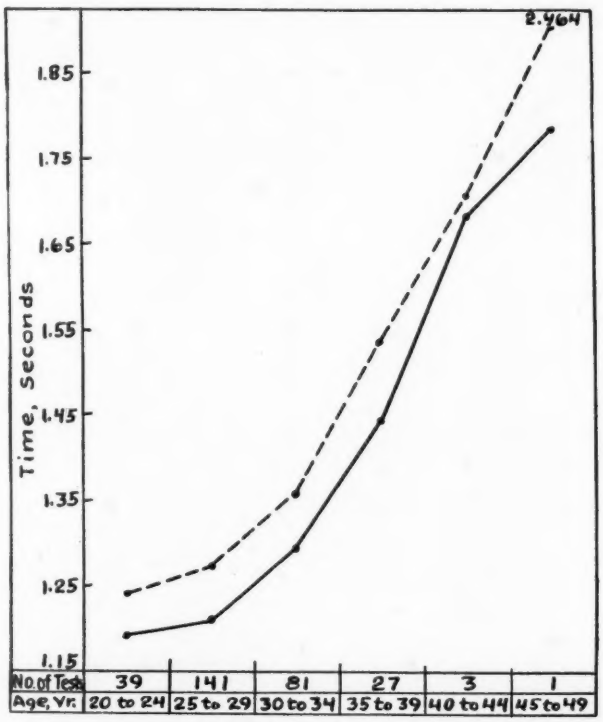

Chart 5.—Speed of accommodation from near to far and return to near of pilots with fatigue after from one to four hours of flying (broken line) compared with their normal speed of accommodation (unbroken line), in terms of the average time for the five year age groups.

Tables 9, 10 and 11 show the average speed of accommodation from near to far, from far to near and from near to far and return to near, respectively, in seconds or fractions of seconds of pilots under normal conditions and pilots with fatigue after one, two, three and four hours of flying compared with their normal speed of accommodation in terms of the average time for the 5 year age groups. There is noted at the bottom of each table the average time for all the age groups under each type group.

TABLE 10.—*Speed of Accommodation from Far to Near of Pilots and Nonpilots and of Pilots with Fatigue After One, Two, Three and Four Hours of Flying and Under Normal Conditions, in Terms of the Average for Each Age Group*

Age, Years	Speed of Pilots, Sec.	Number of Tests	Speed of Nonpilots, Sec.	Number of Tests	Speed After 1 Hr. of Flying, Sec.	Number of Tests	Normal Speed	Speed After 2 Hrs. of Flying, Sec.	Number of Tests	Normal Speed	Speed After 3 Hrs. of Flying, Sec.	Number of Tests	Normal Speed	Speed After 4 Hrs. of Flying, Sec.	Number of Tests	Normal Speed	Speed After from 1 to 4 Hrs. of Flying, Sec.	Number of Tests	Normal Speed	Total Number of Tests for Age Group
20 to 24	0.475	90	0.548	53	0.555	31	0.511	0.493	8	0.485	.....	..	.....	.....	..	.....	0.524	39	0.498	221
25 to 29	0.503	305	0.535	21	0.551	81	0.512	0.542	52	0.504	0.525	4	0.464	0.536	4	0.484	0.538	141	0.491	608
30 to 34	0.538	157	0.623	22	0.551	30	0.519	0.564	30	0.536	0.606	15	0.562	0.590	6	0.509	0.577	81	0.531	341
35 to 39	0.604	57	0.814	5	0.707	10	0.627	0.712	10	0.628	0.649	5	0.573	0.660	2	0.597	0.682	27	0.606	116
40 to 44	0.771	14	0.699	10	0.704	1	0.880	0.968	1	0.770	.....	..	.....	0.946	1	0.770	0.806	3	0.806	30
45 to 49	1.188	5	0.770	1	1.210	1	0.880	.....	..	.....	.....	..	.....	.....	..	.....	1.210	1	0.880	8
Total.....		628		112		154			101			24			13			292		1,324
Total aver.	0.679		0.664		0.713		0.654	0.655		0.584	0.594		0.533	0.683		0.589	0.648		0.590	

TABLE 11.—*Speed of Accommodation from Near to Far and Return to Near of Pilots and Nonpilots and of Pilots with Fatigue After One, Two, Three and Four Hours of Flying and Under Normal Conditions, in Terms of the Average for Each Age Group*

Age, Years	Speed of Pilots, Sec.	Number of Tests	Speed of Nonpilots, Sec.	Number of Tests	Speed After 1 Hr. of Flying, Sec.	Number of Tests	Normal Speed	Speed After 2 Hrs. of Flying, Sec.	Number of Tests	Normal Speed	Speed After 3 Hrs. of Flying, Sec.	Number of Tests	Normal Speed	Speed After 4 Hrs. of Flying, Sec.	Number of Tests	Normal Speed	Speed After from 1 to 4 Hrs. of Flying, Sec.	Number of Tests	Normal Speed	Total Number of Tests for Age Group
20 to 24	1.171	90	1.265	53	1.284	31	1.231	1.197	8	1.150	.....	..	.....	.....	..	.....	1.240	39	1.190	221
25 to 29	1.239	305	1.327	21	1.287	81	1.244	1.287	52	1.251	1.231	4	1.121	1.242	4	1.234	1.274	141	1.212	608
30 to 34	1.329	157	1.380	22	1.303	30	1.296	1.339	30	1.271	1.380	15	1.321	1.361	6	1.286	1.345	81	1.293	341
35 to 39	1.421	57	1.625	5	1.507	10	1.443	1.585	10	1.444	1.575	5	1.470	1.485	2	1.424	1.538	27	1.445	116
40 to 44	1.668	14	1.676	10	1.628	1	1.925	1.892	1	1.595	.....	..	.....	1.683	1	1.590	1.701	3	1.686	30
45 to 49	2.323	5	1.782	1	2.464	1	1.782	.....	..	.....	.....	..	.....	.....	..	.....	2.464	1	1.782	8
Total.....		628		112		159			101			24			13			292		1,324
Total aver.	1.691		1.509		1.578		1.492	1.450		1.341	1.396		1.430	1.492		1.370	1.456		1.408	

The findings for one hundred and twelve nonpilots have been added in these tables merely for the sake of interest and comparison.

Table 8 shows the speed of accommodation from near to far and return to near of pilots with fatigue after from one to four hours of flying compared with their normal speed of accommodation. The effect of fatigue is readily noted in the time and age groups and in the average time of each five year age group.

No consideration is given here to the cases in which the speed was below the limit of safety, as they were incorporated under the separate demonstrations of the speed of accommodation of pilots with fatigue after one, two, three and four hours of flying.

The same precautions were taken in these tests as in previous tests, as noted in my other articles.[1] The illuminometer was used to check illumination and the tachometer to check the speed of the motor. All the tests were carried out by one technician, so that there was no variation in the technic.[1b]

SUMMARY

In previous articles [1] and in the beginning of this article an attempt has been made to establish a standard of speed within safe limitations in five year age groups of the three phases of adjustment of the eye to near and far vision with particular reference for use in aviation. The reasons, including age, for the speed of accommodation's being outside these limitations have been demonstrated.

In the latter part of this article the effect of fatigue is demonstrated, and tables showing the speed of accommodation under normal conditions compared with the speed of accommodation in the fatigued state are given.

CONCLUSIONS

Age has a marked bearing on the speed of adjustment of the eye to clear seeing at different distances.

With any anomalous conditions of the eye the factor of age becomes of greater importance.

After the age of 30 the change becomes rapidly more marked.

Tables 1, 2 and 3 demonstrate what I consider the limits allowable in the three phases of the adjustment of the eye—that from near to far, that from far to near and that from near to far and return to near.

In tables 1, 2 and 3 are demonstrated the variations of the speed of adjustment.

Various anomalous conditions of the eye such as were noted in the discussion of the tests that demonstrated speeds below the limits of safety are, in part, the cause of the slowing of the speed.

Aviators with speed of adjustment not within the limits of safety should be disqualified physically until such time as adjustment can be made, or permanently if it is found that they cannot make the adjustment.

Fatigue has a marked bearing on the speed of adjustment in all ages, more notably so as age progresses.

Fatigue other than that caused by flying can be tested by this method.

The study of fatigue and of the time of recovery is an important phase of this work as yet untouched.

ANTERIOR CAPSULAR CATARACT

AN EXAMPLE OF TRUE METAPLASIA

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The particular nature of the so-called anterior capsular cataract has never been exactly defined. Regarding its pathogenesis, there exists less difference of opinion. As the name indicates, proliferation of the cells belonging to the anterior epithelium of the lens has been generally accepted as the essential factor in the formation of the anterior capsular cataract. However, the nature of the tissue formed from this proliferation of the epithelial cells has been only suggested.

Beer [1] in 1817 described the clinical appearance of anterior capsular cataract. Müller [2] in 1857 discussed the histologic changes and concluded that the new-formed layers show transition to masses of anomalous structure, for example, fibrous tissue. The English pathologists Parsons [3] and Collins and Mayou [4] described the tissue forming the anterior capsular cataract as being composed of flattened epithelial cells and their processes. Hess [5] mentioned only the proliferation of the epithelial cells in the histologic observations. Jess [6] stated that the proliferated epithelial cells are separated from the capsule by many layers of a structure resembling connective tissue. Peters,[7] from the study of a number of cases of anterior capsular cataract, concluded that there exists a true epithelial capsular cataract and that organized connective tissue occurs inside the capsular sac only when it proliferates through a rupture in the capsule of the lens.

Axenfeld,[8] while discussing the possibility of the transition of proliferated pigmented retinal epithelium into connective tissue, wrote that

1. Beer, G. J.: Lehre von den Augenkrankheiten, Vienna, Heubner & Volke, 1817, vol. 2, p. 289.

2. Müller, H.: Ueber die anatomischen Verhältnisse des Kapselstaars, Arch. f. Ophth. **3**:55, 1857.

3. Parsons, J. H.: The Pathology of the Eye, New York, G. P. Putnam's Sons, 1905, vol. 2, p. 407.

4. Collins, E. T., and Mayou, M. S.: Pathology and Bacteriology of the Eye, Philadelphia, P. Blakiston's Son & Co., 1925, p. 596.

5. Hess, C., in von Graefe, T., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, ed. 3, Leipzig, Wilhelm Engelmann, 1911, pt. 2, chap. 6, p. 217.

6. Jess, A., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 5, p. 228.

7. Peters, A.: Zur Kenntnis des Kapselstaars, Arch. f. Ophth. **105**:154, 1921.

8. Axenfeld, T.: Retinitis externa exudativa mit Knochenbildung im sehfähigen Auge, Arch. f. Ophth. **90**:452, 1915.

in the case of proliferations of the epithelium of the capsule of the lens and in the case of capsular cataract, fibrous masses arise which present the characteristics of connective tissue by Van Gieson's and other methods of staining.

In a large collection of human eyeballs anterior capsular cataract is sufficiently common to permit the study of all stages of its formation. From a histologic examination of more than fifty lenses with this condition, I am convinced that anterior capsular cataract is composed of collagenous connective tissue, the fibroblasts of which are directly derived from the epithelial cells of the lens.

The microscopic observations on two patients presenting this change from epithelial cells to fibrous tissue will be briefly described.

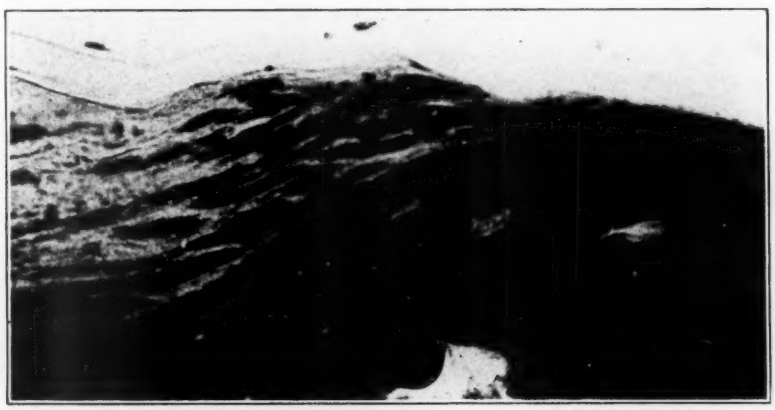
OBSERVATIONS IN TWO CASES

CASE 1.—A man 51 years old noted the loss of all vision in his eye following a spell of coughing. When first examined at the ophthalmic clinic of the Washington University School of Medicine about one month later, this eye presented diminished tension, tremor of the iris and lens, a small degree of clouding in the lens and a grayish reflex from the fundus. Secondary glaucoma developed, making enucleation necessary after nine months because of severe pain. Examination of sections through the enucleated eye confirmed the clinical diagnosis of retinal detachment and dislocation of the lens.

The lens was rounded and presented a diffuse irregularly arranged liquefaction of its cortex. In the middle of the anterior surface of the lens there was observed a small, thin anterior capsular cataract. The part of the capsule of the lens covering the latter was much wrinkled, and on its anterior surface lay a thin layer of posterior synechial tissue and pupillary membrane. From the margin of the pupil adherent to the lens pigmented epithelium from the iris had proliferated for a short distance onto the anterior surface of the pupillary membrane. Since this thin layer of connective tissue on the anterior capsule did not extend beyond the limits of the capsular cataract, it may be concluded that the latter was probably due to the membrane on the surface of the capsule of the lens.

Under the wrinkled lens capsule over the anterior capsular cataract a small amount of albuminous fluid lay in the angles formed by the wrinkles. An occasional thin disk-shaped cell was observed in this fluid. The capsular cataract proper was composed of many thin lamellae arranged with their flat surfaces in a frontal plane or parallel to the anterior surface of the lens. Between these lamellae lay numerous long, thin, flattened spindle-shaped cells having thin, dark-staining nuclei. When flat sections or sections parallel to their flat surfaces were made through these cells, they showed elongated, light-staining cytoplasm with generally oval nuclei. The cells, as a rule, presented two to three short processes of the cytoplasm. These cells in every way resembled mature fibroblasts. When the thin lamellae forming the capsular cataract were stained with Mallory's aniline blue or Van Gieson's trinitrophenol-acid fuchsin stain, they were colored blue with the first stain and red with the second. In other words, these lamellae stained exactly the same as any connective tissue.

At the sides or periphery of the capsular cataract the anterior epithelium of the lens presented considerable proliferation of its cells. Just peripheral to the limits of proliferation of the epithelium of the lens the cells of this single layer of epithelium were considerably flattened. The extra amount of flattening here was probably due to the swelling of the fibers of the cortex of the lens accompanying the diffuse cataractous changes. When the epithelial cells became fibroblasts, the cell body, with the nucleus, which was previously of a uniform although attenuated thickness, flattened to a thin disk with pointed edges. All stages in this transition could be studied at the periphery of the capsular cataract. The junction between proliferated epithelial cells and fibrous tissue might or might not be distinct. Posteriorly, behind the fibrous tissue, proliferation of the epithelial cells extended for a short distance toward the sagittal axis of the lens. A layer of epithelial cells lay neither anterior nor posterior to the connective tissue forming the capsular cataract. The latter lay directly on coagulated detritus resulting from liquefaction of the fibers of the cortex of the lens.



Meridional section through one side of the anterior capsular cataract showing proliferation of the epithelial cells of the lens and, to the left, new-formed connective tissue.

CASE 2.—In a boy 7 years old a perforating corneal ulcer due to gonococci was followed by a prominent staphyloma. Enucleation of the eye was performed eight months after the perforation of the ulcer. Anatomically, it was found that the lens had retracted a little after the perforation but was connected with the cicatricial tissue replacing the cornea by a thick column of new-formed connective tissue extending through the pupil and spreading out on the capsule of the lens with a broad base. Under the latter the capsule of the lens was much wrinkled. The capsular cataract was thicker where the connective tissue lay on the outer surface of the capsule, but on one side the cataract extended for a considerable distance with the capsule over it less wrinkled and uncovered by connective tissue. The cortex of the lens generally presented swelling and liquefaction of the fibers.

On the side of the capsular cataract where no connective tissue lay on the surface of the capsule proliferation of cells from the anterior epithelium of the lens could be studied best. The cells in the single layer of lenticular epithelium peripheral to the margin of the capsular cataract were not so flattened as in the

first case. Consequently the difference in thickness between these epithelial cells and their derivatives the fibroblasts was more pronounced. In a fairly uniform manner the proliferated cells from the epithelium of the lens gradually changed to fibroblasts as one followed them toward the sagittal axis of the lens. In some sections proliferation of the epithelial cells had produced cytoplasmic masses with from two to several nuclei. As in the first case, this proliferation of epithelial cells proceeded behind the capsular cataract for a short distance toward the sagittal axis of the lens. On the opposite side of the capsular cataract, where the much wrinkled capsule was covered by a thick mass of new-formed connective tissue, the same change from epithelial cells to fibroblasts was going on, but here the intermediary cells were not so clearly identified. The thickest part of the capsular cataract lying under the thick deposit of connective tissue on the outer surface of the capsule presented in its anterior two thirds very thick lamellae swollen and in part disintegrated; a few flattened cells were scattered here between the layers. All the posterior lamellae of the thicker part, as well as the lamellae of the thinner part of the capsular cataract under the naked capsule of the lens, were thin, with numerous interlamellar cells. The layers of the capsular cataract were all arranged frontally or parallel to the anterior surface of the lens. A layer of epithelium was absent from either in front of or behind the laminated structure. All the lamellae colored blue with Mallory's aniline blue and red with Van Gieson's stain, which definitely indicated their composition of connective tissue. The interlamellar cells had the flattened disk-shaped appearance with processes characteristic of fibroblasts.

COMMENT

In neither case were there encountered the young forms of fibroblasts having vesicular rod-shaped nuclei and rounded ends, which are seen commonly in granulation tissue. A union of the processes from adjacent fibroblasts could be observed in the second case in flat or frontal sections through the capsular cataract. In the first case the cell bodies did not stain uniformly well enough in the flat sections to determine anything regarding this syncytial condition. The surface view of the fibroblasts in both cases showed the nuclei to be generally oval, light-staining and having one nucleolus. With Verhoeff's stain for elastic tissue, elastic tissue proved to be entirely absent in the connective tissue of the capsular cataract.

Other examples could be described, but to do so would simply be to repeat what has already been said regarding the two cases cited. In cases of capsular cataract that are of long standing, however, all connection with, or evidence of, proliferation in the epithelial cells of the lens has usually departed.

SUMMARY AND CONCLUSIONS

The clinical, anatomic and histologic observations in two cases of recent anterior capsular cataract are described. At the periphery of the capsular cataract in each case reported, proliferation of the cells of the anterior epithelium of the lens, with transition of these proliferated cells

to typical fibroblasts, was observed. Observations confirming the fact that the capsular cataract was composed of collagenous connective tissue are offered.

Since the epithelium of the lens is pure ectodermal epithelium in origin, the formation of connective tissue in capsular cataract is an example of true metaplasia. The designation of capsular cataract is therefore well taken, since the capsular cataract, like the capsule of the lens (Seefelder[9]), is formed from the epithelium of the lens.

9. Seefelder, R., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 1, p. 499.

THE GOLD BALL IMPLANT

SOME ESSENTIAL FEATURES IN OPERATIVE TECHNIC

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The use of implants after enucleation has occupied the attention of ophthalmic surgeons for more than fifty years. A variety of substances have been tried, such as fat, cartilage, bone, ivory, celluloid, paraffin, peat and agar-agar. With the discovery of its shortcomings, each of these has been successively discarded. The material of choice is now a gold or glass ball. The gold ball is generally regarded as the preferred implant. Though in this article I shall discuss the gold ball implant, the technic described applies equally to any form or variety of implant.

In my early experience with implants I paid as little attention to the details of the technic of implantation as does the average writer on the subject. My efforts were crowned with failure and escape of the implant in about half of the cases. These disappointing results led me to study carefully the part played in the unsuccessful attempts by various factors in the technic of the operation.

The first deleterious factor in technic was found to be the use of too large a ball. At the suggestion of Dr. Arthur Chambers, I began to use a sphere not greater than 14 mm. in diameter. Such a sphere was found to fill Tenon's capsule without causing undue tension on the delicate capsular tissue. The successful results increased in number following this procedure.

It was then found that failure to control meticulously any oozing of blood within Tenon's capsule was directly responsible for some failures. Attention paid to this phase of the operation further increased the percentage of successes.

The manner in which Tenon's capsule was sutured was then considered. It appeared that retention of the implant would possibly be better if the edges of Tenon's capsule were overlapped rather than if the margins were merely approximated and sutured with interrupted sutures. The successes following overlapping of the margins proved that this factor in technic was of high significance.

It is impossible to conceive of any success attained by merely suturing the conjunctiva without regard to Tenon's capsule as described by some authors. It is absolutely essential that Tenon's capsule be separately and carefully sutured over the implant.

My present technic has given consistent retention of the gold ball in the last ten cases over a period of five months. The procedure is as follows:

For local anesthesia, a few drops of a 4 per cent solution of cocaine hydrochloride is placed within the conjunctival sac. The usual circumcorneal incision is made. To conserve all the conjunctival tissue possible and to have a cleancut incision, I first pass the point of a knife around the limbus, as suggested by Dr. Edwin Munson, and proceed to undermine the conjunctiva back to the equator. At this stage I inject not more than 1 cc. of a 4 per cent solution of procaine hydrochloride, directing the needle along the outer orbital wall for a distance of 3 cm., with the point directed slightly upward. Here the three branches of the ophthalmic division of the trigeminal nerve enter the orbit through the sphenoidal fissure. These branches and the ciliary ganglion are blocked; this gives sufficient anesthesia for the entire operation.

Each of the four rectus muscles is successively caught on muscle forceps and sutured with 000 catgut to the conjunctiva. Double-armed sutures are passed from the under surface of the muscle through the conjunctiva about 5 mm. from the cut margin. This allows the muscles to assume approximately the same position as in the living eye. The ends of the catgut sutures are left long, held by an artery forceps and laid to one side. The globe is proptosed and cut away with as little loss of tissue as possible. A large spindle-shaped mass of cotton made moist with hot saline solution is packed into the socket until hemorrhage is completely controlled. A dry packing is placed in the socket and kept there to dry the cavity further while one proceeds with the suturing of the edges of Tenon's capsule.

One margin of Tenon's capsule is grasped with a tissue forceps, and a double-armed 000 chromatized catgut suture is introduced from below at a point 2 mm. back of the center of the cut margin of the capsule. This suture is carried to the opposite margin and introduced from below in a similar manner. The suture is left untied for the moment. Two or three additional sutures placed on each side of this one at intervals of 2 mm. are similarly dealt with. The packing is now removed, and a dry cavity will be found. The gold ball is put into place, the sutures are tied, the center one first and then those on each side. The margins of the capsule overlap. If additional sutures are required to close the cavity completely, they can be introduced. A running catgut suture may then be placed in the capsule for reenforcement. The conjunctival wound is closed with no. 2 braided silk.

The four catgut sutures, each of which fixed a rectus muscle to the conjunctiva and was laid to one side at the outset of the operation,

are brought in apposition and loosely tied. Insertion of petrolatum or, preferably, ointment of scarlet red, into the conjunctival space, and a dressing complete the operation. The silk sutures are removed after one week, and at this time the superficial catgut sutures are easily picked off.

My experience indicates that four features in the technic of implantation of the gold ball are essential for success.

1. Tenon's capsule must be sutured separately over the implant. It seems needless to stress this point were it not for the fact that it is omitted by some operators.

2. Oozing of blood in the cavity of the capsules must be carefully controlled.

3. The ball must be small, not greater than 14 mm. in diameter.

4. Closure of Tenon's capsule by overlapping of the margins is essential.

CORALLIFORM CATARACT AND A NEW FORM OF CONGENITAL CATARACT WITH CRYSTALS IN THE LENS

SANFORD R. GIFFORD, M.D.
AND
IRVING PUNTENNEY, M.D.
CHICAGO

The name coralliform cataract was first applied by Gunn [1] to a particular type of congenital cataract in which many layers of the lens in the axial area are affected by peculiar branching opacities. Related forms of opacity which have this peculiarity of distribution have been called floriform, arborescent and fusiform cataract, while the form described by Vogt [2] as spear cataract also belongs in this group. In the latter's cases the opacities were composed of shiny crystals arranged about denser central rods to form a series of spear-shaped figures joined together at irregular angles more or less near the sagittal axis of the lens. The opacities gave the impression of a number of spiny insects or caterpillars in the axial area. A mother, a son, a maternal aunt and two cousins were affected. In 1924 one of us [3] reported a case resembling that of Vogt in which similar crystals were arranged more regularly in the axial area so as to resemble two fir trees with their bases together in the center of the lens, the tips coming nearly to the anterior and the posterior capsule. The only other cases of a condition designated by the name spear cataract which we have found in the literature are those of two sisters, reported by Cords,[4] though the relation between this form of opacity and the forms described by other names is so close that sharp differentiation is in many cases impossible. In 1927 one of us [5] reported two cases of the more common form, which could be described as coralliform. The literature on the few reported cases

From the Department of Ophthalmology, Northwestern University Medical School.

1. Gunn, R. M.: Tr. Ophth. Soc. U. Kingdom **15**:119, 1895.

2. Vogt, A.: (*a*) Arch. f. Ophth. **107**:446, 1922; (*b*) Lehrbuch und Atlas der Spaltlampen Mikroskopie des lebendes Auges, Berlin, Julius Springer, 1931, pt. 2, p. 446.

3. Gifford, S. R.: Am. J. Ophth. **7**:678, 1924.

4. Cords, R.: Klin. Monatsbl. f. Augenh. **76**:125, 1926.

5. Gifford, S. R.: Klin. Monatsbl. f. Augenh. **78**:194, 1927.

in these groups will be found in these papers. In nearly all the cases the opacities were bilateral, and often several members of a family were affected. In the family of Gunn's patient Nettleship found nineteen cases of typical coralliform opacities. In the family studied by Knies, anterior and posterior polar cataracts connected by a threadlike opacity to a lamellar opacity near the center of the lens were present in two members, while two other members showed anterior polar and nuclear opacities.

Apparently in the whole group of cases the lesion first appears in early fetal life, affecting the region of the embryonal nucleus. Layers of cortex of later development are each affected in the central area only, the extent of opacity being often less in the succeeding layers till, as in my cases, a layer next to the capsule may be completely spared.

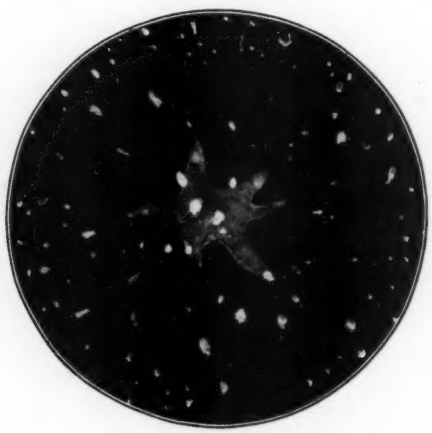

Fig. 1.—Opacities in the lens of the right eye in case 1.

We are reporting an additional case of coralliform cataract and also a case of congenital cataract of even more unusual form, observed within the past three years, in which it was possible to investigate the character of the lenticular opacities by microchemical methods. The investigation was fairly complete in case 2 only, because of insufficient material in case 1.

REPORT OF CASES

CASE 1.—Mrs. J., a woman of 28, had first noticed poor vision at the age of 18. Vision rapidly became worse, so that four years later she was unable to read. There was a history of cataract in the family. The mother was told at 32 that she had "blue cataract." When she was seen by one of us at the age of 50, however, the cataracts were far advanced and could not be distinguished from senile cortical cataract. In a maternal uncle cataract developed at the age of 55. The patient has two older sisters and one brother who know of no ocular defect.

When the patient was first seen vision was 4/200 in the right eye and 20/100 in the left. The right eye showed opacities of the lens as illustrated in figure 1.

The irregular stellate or flower-shaped opacities extended forward to the anterior capsule and backward to the posterior capsule, being connected with similar branching opacities involving all layers of the lens in the axial area. Outside this area the cortex contained many fine punctate and larger flaky opacities; as seen with the slit lamp these gave a play of colors from green to purple. The left eye showed a similar picture except that the branching axial opacities were not so extensive. The opacity in the anterior capsule was limited to a small oval area.

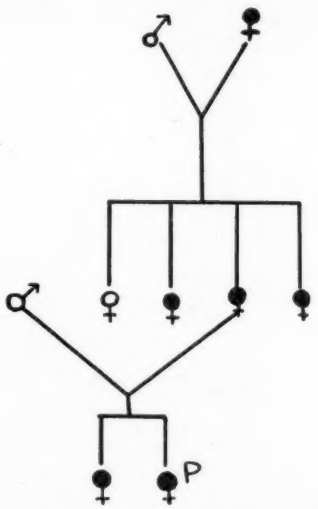

Fig. 2.—Family tree of the patient in case 2. The black symbols indicate affected patients.

Fig. 3.—Opacities in the lens of the left eye in case 2.

Within a period of six months both lenses were removed by corneal expression after preliminary discission, with resulting vision of 20/15 in each eye. The expressed material was collected and examined microscopically. The fresh moist material showed long needle-shaped crystals somewhat resembling those seen in case 2, but no photomicrograph was taken. The crystals were insoluble in chloroform and a 70 per cent solution of alcohol. They were at least relatively insoluble in

cold water, since their shape was preserved in moist preparations. Acids and alkalis were not tried, nor were any chemical determinations made. Insolubility of the crystals in chloroform, ether and alcohol apparently excludes cholesterol and other lipoids, so far as the larger crystals are concerned. Millon's reaction was negative, so it was thought that the protein crystals described by Verhoeff could be excluded.

Case 2.—Miss V., aged 20, whose vision had been poor since infancy, had undergone needling of the right eye at the age of 13. Owing to the death of the operator

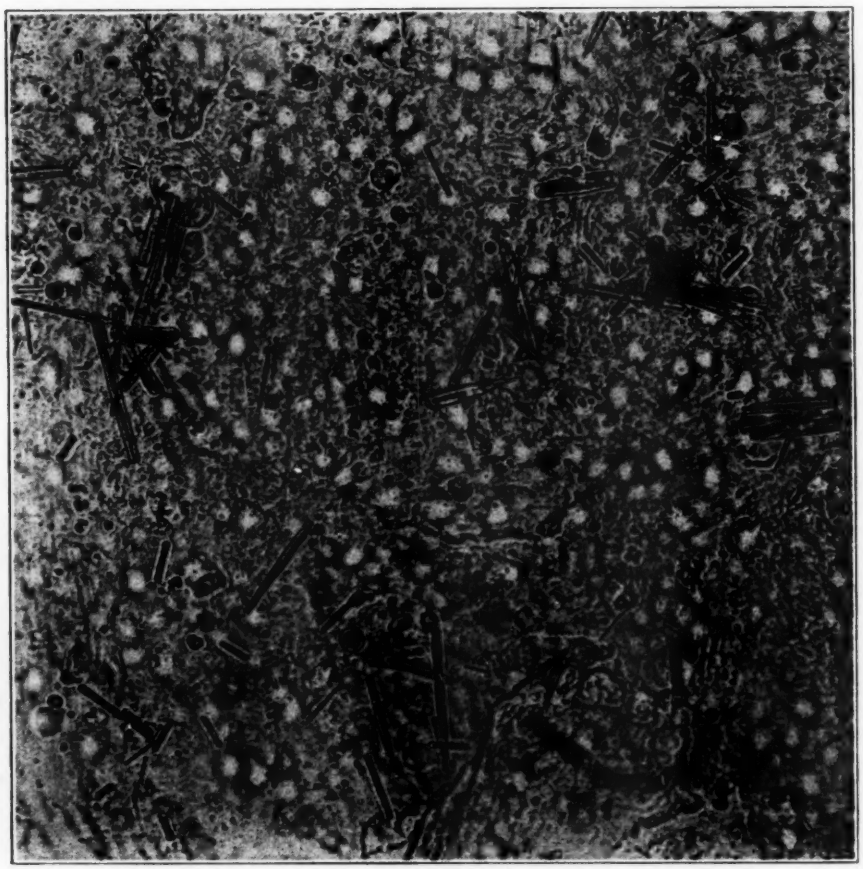

Fig. 4.—Photomicrograph of crystals of the opacity in case 2.

not long afterward, no subsequent operations were done, and she had attended the Indiana School for the Blind.

The patient knew that congenital cataract was common in the maternal side of her family, the mother, maternal grandmother and four maternal aunts being affected, while one maternal aunt was spared. The patient's only sister was affected (fig. 2). The patient had no brothers. The two maternal aunts had previously been cared for by Dr. H. Gifford and one of us. One aunt at the age of 18 months showed what were described as zonular cataracts with several distinct yellowish spots in the nuclei. Vision of 20/70, as found fifteen years later,

was obtained after discissions. The other materal aunt, who was seen at the age of 37, showed "very dense lamellar cataracts, about 6 mm. in diameter." No mention of crystals is made in the record. Vision of 20/25 and 20/30 was obtained after discission and corneal expression.

Vision of the right eye of our patient was 15/70, and that of the left eye was 20/100, with correction. The right eye showed a fairly dense membrane, with a small opening and apparently no cortex remaining. The left eye showed the condition illustrated in figure 3. The opacity was composed of crystals which resembled those seen in the case of spear cataract but were arranged in a more compact and circular mass. This mass could be divided into an outer zone measuring 6 mm. in diameter and an inner one measuring 3 mm. The space between these zones

Fig. 5.—Photomicrograph of crystals of calcium sulfate.

also contained crystals, but these were more sparsely distributed. The small inner mass was localized by the anterior Y suture which rested on its anterior surface. A thin layer of normal cortex was present anterior to the outer zone of crystals. Presumably it extended to the posterior cortex, but this could not be seen. The crystals were needle shaped and of various sizes. They were not so shiny as those seen in case 1, and no play of color was observed.

Obviously this picture did not resemble that of case 1, nor was it like that of Vogt's spear cataract. While the concentric zones of denser opacity suggested a development similar to that of zonular cataract with involvement of successive layers of cortex, the fact that the whole opacity consisted of crystals distinguished the picture definitely from that of ordinary zonular cataract. The arrangement can perhaps best be compared with the zones of light seen in a revolving pinwheel.

I have not found descriptions of its exact counterpart in the literature. In the cases described by Vogt [6] the eyes presented some points in common, especially in one case, in which there were "rosette-like concentric club-shaped opacities," but in none was there any close resemblance.

In the right eye a cut through the membrane by Wheeler's method produced an opening through which vision of 20/40 was obtained with correction. In the left eye a preliminary discission was done, followed in three days by corneal expression of the soft cortex. The central mass of opacities remained in a fairly coherent isolated mass and was collected in a large pipet for immediate examination. Some cortex remained which absorbed slowly; vision was 20/50 three months after operation.

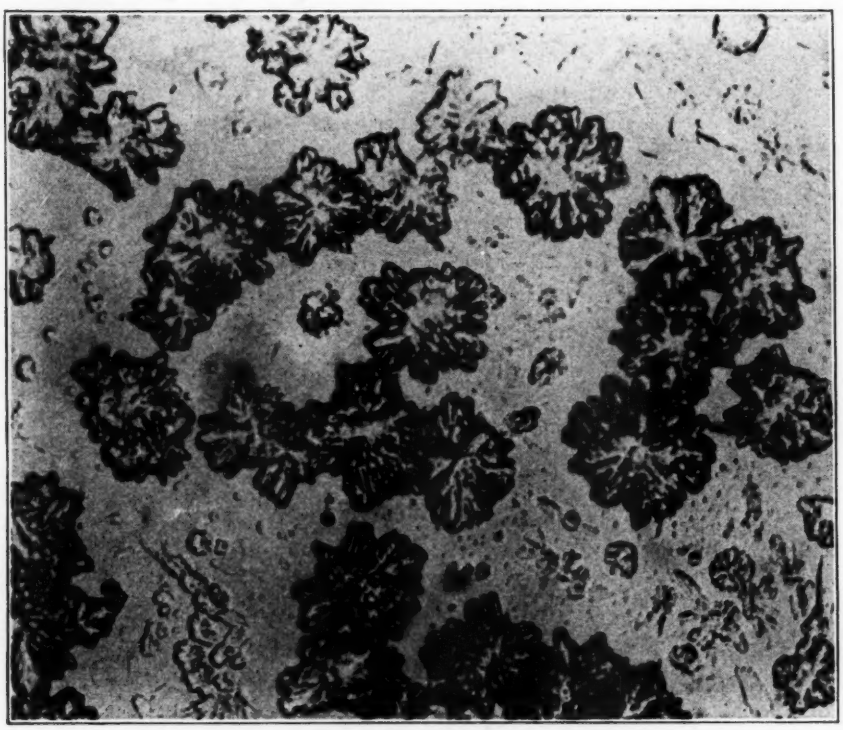

Fig. 6.—Photomicrograph of crystals of calcium phosphate.

Examination of the soft material containing the opacity showed the crystals illustrated in figure 4. These were long polyhedral needles of fairly regular width but varying in length. They resembled most closely the needle-like crystals of calcium sulfate (fig. 5). Some crystals of calcium phosphate are shown in figure 6 for comparison.

The crystals were insoluble in alcohol, ether, glycerin, dilute ammonium hydrate and hot and cold water. They were soluble in dilute sulfuric acid. Insolubility in ether excluded lipids and cholesterol as constituents of the crystals. They

6. Vogt,[2b] pp. 429, 433 and 444.

resembled alanine crystals in form, but this and other amino-acids were excluded because of insolubility in hot and cold water. Solubility in dilute sulfuric acid made it appear almost certain that they contained a calcium compound.

In view of this the remaining material was employed for a quantitative determination of calcium. This showed 0.6 per cent of calcium in the moist material.

COMMENT

According to Salit,[7] the normal lens contains no calcium, while Adams [8] found 0.0185 per cent. In lenses with incipient cataract Salit found 0.0058 per cent, and in those with mature senile cataract, 0.0509 per cent, the greatest amount in any lens being 0.108 per cent. Adams found 0.0653 per cent in lenses with cataracts of various types. Hence the amount of calcium found in the opaque portion of our lens was ten times the average amount found in cataractous lenses by Adams, which offers additional proof that the crystals examined were composed chiefly of calcium.

The presence of crystals in the cataractous lens is not uncommon. In the cataract of patients with myotonic dystrophy crystals are nearly always observed and from their flaky form and the play of colors observed with the slit lamp have usually been considered as cholesterol crystals. A few recent reports by Cattaneo,[9] Kranz [10] and others have shown that by means of examination by polarized light the presence of cholesterol, other lipids and calcium may be determined in the cataractous lens before operation. Kranz found that 30 per cent of normal lenses showed some crystals, chiefly lipid crystals, by this method, while in cases of the common coronary cataract crystals were found in 80 per cent. He did not examine any lenses resembling those here described.

Cases of congenital cataract showing crystals which have been examined microchemically are few in number. Braun [11] examined with especial care the lenses of a woman with cataract greatly resembling the coralliform type. A son was affected with identical opacities in the otherwise clear lens. Braun found the crystals to be those of a compound of tyrosine. Calcium and cystine were also found but were not considered as principal constituents of the crystals. Vogt found in one of his cases of spear cataract crystals which consisted chiefly of cysteine, though cholesterol could not be excluded. Beresinskaja [12] described

7. Salit, P. W.: Am. J. Ophth. **13**:1072, 1930; Calcium Content and Weight of Human Cataractous Lenses, Arch. Ophth. **9**:571 (April) 1933.

8. Adams, D. R.: Biochem. J. **23**:902, 1929.

9. Cattaneo, D.: Ann. d'ocul. **165**:105, 1928.

10. Kranz, H. W.: Klin. Monatsbl. f. Augenh. **79**:654, 1927; Ztschr. f. Augenh. **65**:219, 1928; Arch. f. Ophth. **118**:571, 1927.

11. Braun, G.: Arch. f. Ophth. **118**:701, 1927.

12. Beresinskaja: Klin. Monatsbl. f. Augenh. **80**:125, 1928.

a familial zonular cataract in which crystals could be seen which proved on analysis to contain cholesterol and some traces of cystine. Verhoeff [13] examined carefully the lenses of a man with typical coralliform cataract. The crystals were larger than those seen in our cases and were insoluble in acids, alkalis, alcohol and ether. They were digested by pepsin in a 0.4 per cent solution of hydrochloric acid and gave a positive test with Millon's reagent. They were considered to be a crystalline form of protein like that found in certain plants.

CONCLUSIONS

The reports indicate that various chemical changes may occur in lenses with opacities belonging in the group of coralliform cataract, resulting in the formation of crystals in the lens.

In certain cases the crystals themselves seemed to be chiefly responsible for the opacity. Thus, in the cases studied by Vogt, Verhoeff and Braun and in the second case described here the lenses were clear except for the crystals.

While resembling each other in distribution and in general appearance, on careful slit lamp study the crystals revealed in most cases differences which, in view of the chemical findings in the cases studied, would give some indication of their chemical nature. One is not justified in calling any crystals that give a play of colors cholesterol crystals.

There seems to be no doubt that the crystals in our second case were those of a salt of calcium, probably calcium sulfate, while in the first case it is probable that they were of a similar nature.

That a chemical process which results in the formation of such crystals in the lens should be transmitted by heredity is noteworthy. This is not true in all cases, since in certain families other forms of cataract, not characterized by crystals, occurred. It was noteworthy, however, in the families studied by Vogt and Braun in which identical crystals and no other changes were present in two generations.

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13. Verhoeff, F. H.: Arch. Ophth. **47**:558, 1918.

Clinical Notes

AN AID TO FACILITATE THE OPHTHALMOSCOPIC EXAMINATION

JAMES ASA SHIELD, M.D., RICHMOND, VA.

The ophthalmoscopic examination is made with less effort and therefore with less fatigue on the part of the patient if it is possible to orient oneself immediately. When the eye is so placed that the ophthalmologist looks straight through the pupil at the optic nerve head he can quickly make his study. The purpose of the procedure that I use is to enable the patient to place his eye in the position of choice and to make it easier for him to keep his eye in this position.

One enounters little difficulty in making the ophthalmoscopic examination of many patients when using the routine directions, namely, asking the patient to look straight ahead, to look slightly to the right or to the left, to look at the ceiling or to look at some object. However, the patient may find difficulty in following such directions, especially when the examiner's head necessarily obstructs his view in part or completely. It has been my experience that when the vision of the patient is obstructed by my ophthalmoscope and my head his eye will often wander. The procedure to prevent this has been evolved. I attempt to utilize the patient's sense of position so as to place the eye in the optimal position for ophthalmoscopic examination. In the case of babies I attempt to utilize their sense of hearing and position, and in the case of psychotic persons, to utilize their sense of hearing, their sense of position and their sense of feeling.

The patient is instructed to place his right arm straight out at right angles to the chest and is told to look at his right thumb. (It is usually easier to take the patient's hand and place it in the desired position and tell him to hold it there and look in the direction of his thumb.) The ophthalmologist's head may prevent the patient from seeing his thumb, but he knows where his thumb is and can focus his eye in that direction whether or not he can see it. When the right thumb has been placed forward, straight out from the shoulder and at right angles to the chest, it is in perfect position to place the left eye in the best location for examining the fundus, and it follows that the left thumb so placed fixes the right eye in the correct location for examination. The advantage to the patient, particularly to a nervous patient, is definite when one is able to cut down the time necessary to make the ophthalmoscopic examination. The student who is able to look straight through the pupil at the disk is encouraged to look at more eyes, and the patients who are used for demonstration do not become fatigued as quickly when the time required for this study by the student is markedly reduced.

From the Department of Neuropsychiatry, the Medical College of Virginia.

Certain patients—those with paresis, paralysis, disturbances of consciousness of varying degrees or involvement of attention to various extents—are unable to hold their hands in the correct position; this procedure is of much value in such cases. In these cases I have the nurse hold the patient's hand and ask him to look at his thumb, augmenting his attention by placing in his hand something warm or something cold. This will tend to make the patients place their eyes in the proper position, and though they are mentally disturbed my associates and I have been able to reach their consciousness to the extent of getting brief periods of cooperative effort on their part.

The examination of children is facilitated occasionally by having the nurse hold in their hands a watch or a toy, preferably a toy that will make a noise, such as a small crying doll. In the case of young babies I place a watch by the side of the baby's ear, let him look at the watch, place it back by the side of his ear and then have the nurse hold the watch in his hand, which is held in the correct position. In this way I utilize the patient's attention to his hand, to the toy, to the watch, and to the sound made by the watch or the toy.

The location of the object in the hand when the arm is held at right angles to the chest on a level with the shoulder, or slightly higher, as the examiner prefers, enables the nurse to keep the objects used for attention anchored in the optimal position to fix the eye so that the examination is made with the greatest of ease.

I do not know whether this method has ever been used before or whether it is in general use by some physicians. It is a procedure that I have evolved and found workable for my students and myself.

INSTRUMENTS FOR TREATMENT OF THE LID

PARKER HEATH, M.D., DETROIT

Herewith is submitted a set of instruments for treatment of the lids which save a great deal of time for the surgeon. The instruments are shown in the accompanying illustrations. They are made by V. Mueller and Company.

Fig. 1.—A dilator for the punctum, of convenient size and with a nonslipping handle, which can be used with a sharp needle point or with a duller point.

Fig. 2.—A double-ended instrument for massage and expression of the contents of the meibomian and other glands of the lids.

Fig. 3.—A convenient conjunctival flap dissector, useful especially in operations with the trephine, which enables one to dissect out a pocket between the scleral ingrowths, the favorite site for making a trephine opening.

Fig. 4.—A series of ring curets, with dull and sharp sides, which are easily kept clean and are easy to manipulate, especially in operations for chalazion.

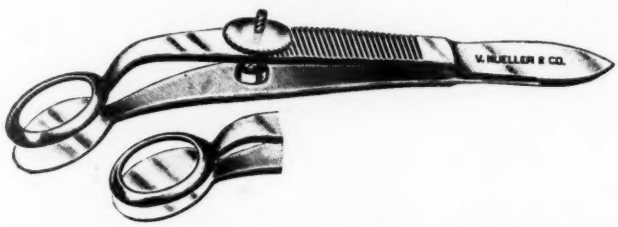

Fig. 5.—A chalazion clamp of the proper shape, one that will not compress the border of the lid.

Fig. 6.—An applicator holder which is easily kept clean and which enables the surgeon to select one applicator easily without contaminating the others.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

EARLY SIMPLE GLAUCOMA

ITS DIAGNOSIS AND MANAGEMENT

FREDERICK C. CORDES, M.D.

SAN FRANCISCO

Glaucoma is a collective term applied to all diseases of the eye the principal symptom of which is increase in the intra-ocular pressure. In certain eyes the intra-ocular tension is above the average normal but causes no functional changes; this condition cannot be called glaucoma.

NORMAL INTRA-OCULAR TENSION

The tension of the normal eye in the vast majority of cases varies between 21 and 27 mm. of mercury. According to P. Knapp,[1] Langenhan [2] and Bruns,[3] this is uninfluenced by sex or refraction, while Guglianetti [4] found that temperature, humidity and atmospheric pressure did not play a rôle.

The intra-ocular pressure is not absolutely constant but shows certain slight variations. The influence of the variations of the blood pressure due to the respiratory rate and the pulse rate, as expressed by the rhythmic oscillations of the pressure, can be seen roughly by the movement of the pointer on the Schiötz tonometer. A second form of deviation is the regular daily variations. Regular tonometer readings over twenty-four hour periods show a daily variation of from 2 to 3 mm., the pressure being higher in the morning than at night. Many causes have been suggested for this, such as the horizontal position in sleep, the absorption of food, failure of the pumping action of accommodation and the absence of muscular movement.

Szymanski and Wladyczko [5] were able to demonstrate experimentally that an excavation of the disk follows a rapid decrease of intracranial

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From the Division of Ophthalmology, the University of California Medical School.

1. Knapp, P.: Klin. Monatsbl. f. Augenh. (pt. 1) **50**:691, 1912.
2. Langenhan, in von Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1904, vol. 4, p. 606.
3. Bruns: Klin. Monatsbl. f. Augenh. **71**:90, 1923.
4. Guglianetti, L.: Arch. di ottal. **21**:382, 1913-1914
5. Szymanski and Wladyczko: Klin. Oczna **3**:145, 1925; abstr., Zentralbl. f. d. ges. Ophth. **16**:729, 1926.

pressure, while Parker [6] was able to produce choked disks by rapid reduction of the intra-ocular tension. Block and Oppenheimer,[7] however, found that there is no apparent relationship between the intracranial pressure and the intra-ocular tension, although both are affected by a change in the osmotic concentration of the blood.

A good deal has recently been written on the relationship of the intra-ocular pressure and the blood pressure, the permeability of the vessels and its effect on the intra-ocular tension. While this work is in the theoretical stage, it seems to be pretty well established that a sudden large variation of the general blood pressure can produce changes in the intra-ocular pressure.

The effect of the glands of internal secretion on the vascular system is well known. That these glands also affect the intra-ocular tension has been brought out by experimentation and clinical observation. Hertel [8] found that extirpation of the thyroid caused an increase of the intra-ocular tension, while feeding thyroid produced a drop in the pressure. These observations were confirmed by A. Fuchs,[9] Lamb [10] and others. Imre [11] observed that in hyperpituitarism the tension was rather low and that pregnant women showed the tendency to low tension. Cucchia [12] found that extirpation of the ovaries had no influence on the tension, while Salvati [13] found a slight increase in the tension during menstruation. Such observations as the aforementioned have led to attempted treatment of glaucoma by organotherapy. From the clinical and experimental observations it seems evident, however, that the endocrine glands do influence tension.

The relation of the intra-ocular tension and nervous influence has long been recognized. Research and clinical observations, such as those of Abadie,[14] have shown that the regulation of the intra-ocular tension is a function of the sympathetic nervous system. Henderson and Starling [15] in 1904 stated that the intra-ocular pressure is a function of the blood pressure in the ocular blood vessels and varies directly as the latter. They demonstrated that the sympathetic nervous system contains

6. Parker, W.: Tr. Am. Acad. Ophth. **29**:77, 1924.
7. Block, E. B., and Oppenheimer, R. H.: A Comparative Study of Intraspinal Pressure, Blood Pressure and Intra-Ocular Tension, Arch. Neurol. & Psychiat. **11**:444 (April) 1924.
8. Hertel, E.: Arch. f. Ophth. **88**:197, 1914.
9. Fuchs, A.: Bull. Ophth. Soc. Egypt., 1924, p. 23.
10. Lamb: Tr. Am. Ophth. Soc. **24**:105, 1926.
11. Imre, J., Jr.: Endocrinology **6**:213, 1922.
12. Cucchia, A.: Ann. di ottal. e clin. ocul. **56**:117, 1928.
13. Salvati: Ann. d'ocul. **160**:568, 1923.
14. Abadie: Clin. opht. **12**:303, 1923.
15. Henderson, E. E., and Starling, E. H.: J. Physiol. **31**:305, 1904.

vasoconstrictor fibers going to the uveal blood vessels and that stimulation causes constriction of the intra-ocular blood vessels. Adler, Landis and Jackson [16] were able to show experimentally that an increase of the blood pressure produces an increase in the intra-ocular tension. The local vasoconstriction through the cervical portion of the sympathetic nervous system, however, acts as a protective measure to prevent sudden harmful increase in the intra-ocular pressure. These authors also found no effect on the intra-ocular tension when the cervical portion of the sympathetic trunk was cut when the blood pressure was normal. After section of the sympathetic trunk, however, there was a higher intra-ocular pressure than in the control animal with an increased blood pressure. Stimulation of the cervical sympathetic nerve fibers shows, as a rule, a temporary reduction of tension in both normal and glaucomatous eyes.

From the brief review of the physiologic features of the intra-ocular tension just given it becomes apparent that many factors have a bearing on its control. This explains the difficulty in understanding the various elements that play a part in the production of primary glaucoma. It is possible that in the future ophthalmologists may have to change completely their conception of glaucoma and may perhaps be able to classify it on an etiologic basis.

GLAUCOMA

At the present time glaucoma, exclusive of the congenital type (hydrophthalmos), is divided clinically into two main groups, primary and secondary. The cases in which glaucoma results from visible pathologic changes in the eye, such as occlusion of the pupil, are known as cases of secondary glaucoma. In contradistinction to secondary glaucoma, there is primary glaucoma. In this type there is no demonstrable disease of the eye that can be regarded as a causative factor.

Primary glaucoma is not a single symptom complex. The different forms are determined by the height of the intra-ocular pressure, the localization and degree of the pathologico-anatomic changes and the resulting disturbance of function. The variation in the picture of the symptom complex caused by these factors suggests a classification of primary glaucoma into the following types: (1) acute inflammatory (acute uncompensated), (2) chronic simple (compensated), (3) chronic inflammatory (chronic uncompensated) and (4) absolute (degenerative). Here I am concerned only with chronic simple glaucoma in its early stages.

16. Adler, F. H.; Landis, E. M., and Jackson, C. L.: Arch. Ophth. **53**:230, 1924.

In glaucoma simplex both eyes are usually involved, and the intra-ocular pressure is, as a rule, consistently higher than in the normal eye. While these eyes show a complete regulation of the production and drainage of aqueous, the fine balance of the pressure-regulating apparatus has been disturbed. A certain balance, however, still persists, so that acute increase of pressure is not present and thus there is no dilatation of the veins. It is because of this lack of congestion that simple glaucoma is also called compensated glaucoma.

In the beginning of the disease the subjective signs are mild or completely absent. Pain, foggy vision and rainbow colors are, as a rule, absent. The persistently increased intra-ocular tension gradually produces excavation and atrophy of the optic nerve. It is only when these factors produce disturbance of vision that the patient seeks medical aid. If the change is gradual the condition may not be observed or may be attributed to senile changes. If one eye is involved the condition may be called to the patient's attention only when some accident makes him close the other eye. Externally the eye cannot be differentiated from the normal eye except by certain detailed minute examinations. The ophthalmoscopic examination and the manual palpation of the eye during a routine examination may reveal signs that lead one to suspect early glaucoma. As Gradle [17] has pointed out, there are also certain hints that may be derived from the history. In a patient in the age period when glaucoma is most prevalent "the story of headaches appearing after a stay of twenty minutes or more in a darkened room, such as at a moving picture theatre, should arouse suspicion. Again, headaches appearing during the morning hours may demand further investigation. In some instances, a digestive upset is the precursor of a simple glaucoma." The presence of glaucoma in the family history should also make one suspicious.

The early diagnosis of simple glaucoma is of great importance and is often difficult. The borderline cases are all too frequently overlooked, and it is for the detection of these cases that the ophthalmologist must be ever on the alert. In cases in which the condition is suspected, in addition to the routine examination a study of the curve for tension and certain provocative tests for compensated glaucoma are indicated. These tests often point to the diagnosis and give the indication for the type of therapy demanded.

INTRA-OCULAR TENSION IN GLAUCOMA

The height of the intra-ocular tension in glaucoma varies a good deal. Most frequently the tension is between 30 and 50 mm. of mercury. There may be instances in which the pressure is higher or lower. There are, on rare occasions, cases in which the pressure is below the upper

17. Gradle, H.: Am. J. Ophth. **14**:936, 1931.

limits of normal and in which all the signs of glaucoma are present. These are referred to as cases of "glaucoma without increased tension."

In these cases of glaucoma without increased tension, according to A. Knapp,[18] the condition is not glaucoma. While the patient shows atrophy of the optic nerve with excavation of the disk, he does not show increased tension at any time. The perimetric changes have a tendency toward altitudinal defects and do not correspond to the usual defects of the field seen in glaucoma. There is also a tendency to show no progression after a certain stage. The tension is often low. In one of the three cases that I have personally observed the tension never rose above 18 mm. (Schiötz) even when the tension was taken over the twenty-four hour period. Roentgen examination shows calcification of the internal carotid, posterior communicating and ophthalmic arteries, which Knapp feels is the etiologic factor.

The change from normal tension to pathologic tension in glaucoma simplex is gradual and insidious. Sudden changes such as are noted in acute glaucoma do not occur. In the course of years the tension gradually and slowly increases until it reaches a pathologic height. There is, however, a regular daily and yearly variation that is important from a diagnostic and a prognostic point of view. In the understanding of glaucoma a curve for tension is just as important as a curve for temperature in infectious diseases.

DAILY CURVE FOR TENSION

If the tension is taken every four hours during the twenty-four hours of a day, the normal eye will show no appreciable variation (fig. 1). In the glaucomatous eye, on the other hand, there is a definite variation, which may amount to 20 mm. of mercury. The intra-ocular tension reaches its highest point between 5 and 7 a. m. On arising there is a rather sudden drop, the tension remaining lower until late at night. At night the rise takes place in two states—a rather gradual increase until 3 o'clock and then a sudden rise that reaches its high point between 5 and 7 o'clock (fig. 2). According to Thiel,[19] this is the typical curve for glaucoma simplex as shown by the examination of extensive clinical material. This curve has been explained as due to change in the distribution of the blood of the body, the cessation of digestion probably being an important factor. Taken over a period of days the curve for tension shows a consistent daily variation (fig. 3). If both eyes are involved the curves are similar, whereas if only one eye is affected the other will show but little variation. If, however, the apparently normal eye shows a variation of more than 10 mm. this must be regarded as a sign of latent glaucoma.

18. Knapp, A.: Association of Sclerosis of the Cerebral Basal Vessels with Optic Atrophy and Cupping, Arch. Ophth. **8**:637 (Nov.) 1932.

19. Thiel, R.: Arch. f. Augenh. **96**:331, 1925.

From this it appears that in cases in which the signs lead one to suspect early glaucoma, a reading of tension once a day is not sufficient, but the tension should be taken twice a day, once in the early morning

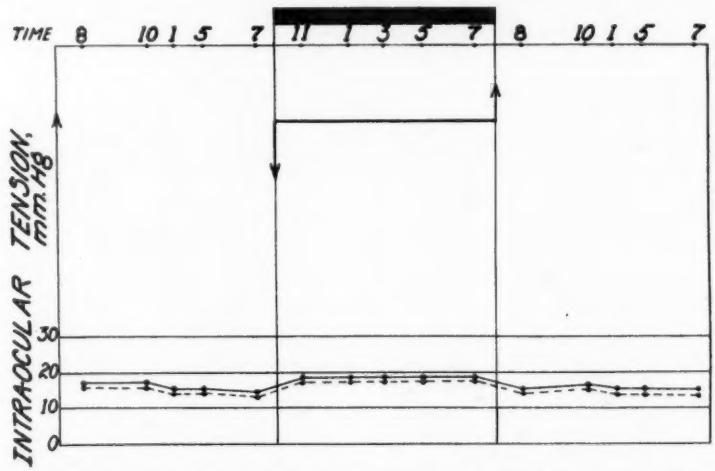

Fig. 1.—Daily curve for tension of a normal right and left eye. Charts 1 to 6 were adapted from Thiel, R., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 4, p. 717.

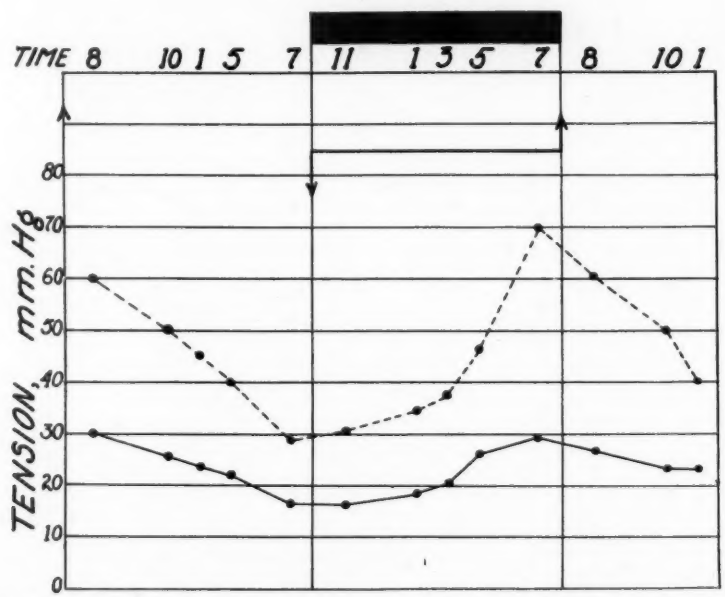

Fig. 2.—Typical curves for tension for two eyes with early chronic simple glaucoma.

and again late in the afternoon. If possible, hospitalization, with taking of the tension during intervals over from three to four days, is preferable. In regard to the early morning tension, it is important to take

this at a time when it is necessary to awaken the patient to take the tension. From a practical point of view it is not necessary to awaken the patient during the early morning hours. Taking the tension every four hours during the day until 11 p. m. and then again at 6 a. m. will suffice to bring out any abnormal curve. In those cases in which the tension remains normal during the day but rises to abnormal heights during the night, the curve for tension is particularly valuable and will explain the presence of early changes in the central field.

If the curve does not show the typical form or if hospitalization is not possible, it is often possible to establish the diagnosis by the so-called provocative tests. The adaptability of the eyes to appreciable variations in the blood pressure and the changes in the distribution of blood form the basis for these tests. While the intra-ocular vascular system

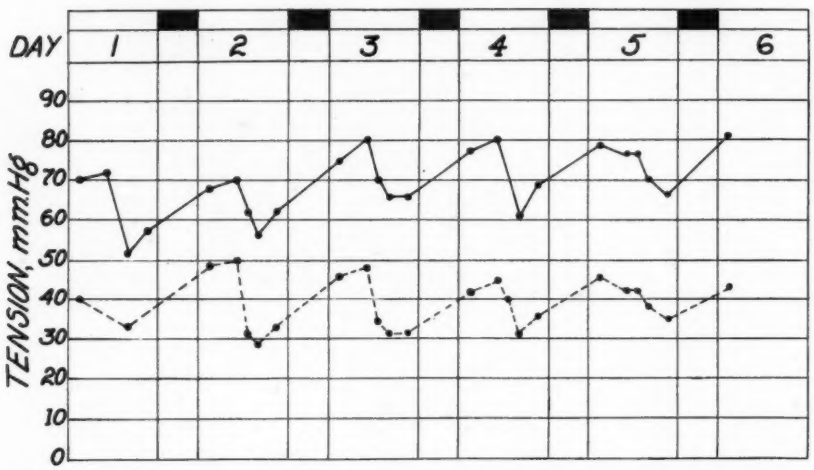

Fig. 3.—Constant daily curve in glaucoma simplex for the right and the left eye.

shows these changes, the tension is unaffected in the normal eye. In the glaucomatous eye this mechanism for regulation is disturbed before other gross functional changes are present.

CAFFEINE TEST

In its simplest form this test is carried out by giving the patient a cup of strong coffee or by giving an intravenous injection of 0.2 Gm. of caffeine. Löhlein,[20] Wegner [21] and others found that in glaucomatous eyes there is an increase of the intra-ocular tension that runs parallel to the increase in the blood pressure, while in the normal eye there is no variation (fig. 4).

20. Löhlein: Klin. Monatsbl. f. Augenh. **77**:909, 1926.
21. Wegner: Zentralbl. f. d. ges. Ophth. **24**:1, 1930.

VENOUS CONGESTION TEST

A tourniquet is placed around the neck just tight enough to cause venous congestion but without interfering with speaking or swallowing. The tension is taken before the procedure and at the end of an hour. An increase of more than from 5 to 6 mm. of mercury speaks for latent glaucoma. As a control, the tension is again taken ten minutes after the tourniquet has been removed. At this time, as a rule, the tension is below that taken before the start of the test. This test should not be applied to old patients or patients with advanced arteriosclerosis (fig. 4).

A simpler way of carrying out this test, according to Wegner,[21] is to place the patient on a table with the head lowered from 20 to 30

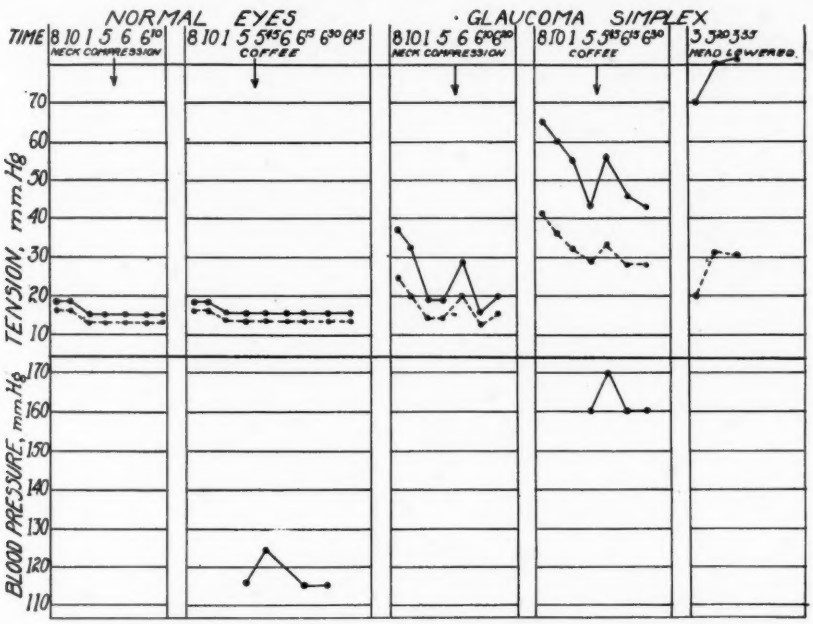

Fig. 4.—Curves for tension of normal eyes and glaucomatous eyes after compression of the neck, the drinking of coffee and lowering of the head.

degrees for from ten to fifteen minutes, when the tension, which has been taken before the test, is again taken. The variations already noted also apply with this method (fig. 4).

MASSAGE

A simple functional test is massage of the eyeball with the tonometer. If in the normal eye the Schiötz tonometer with the 5.5 Gm. weight is kept constantly resting on the eye for from three to five minutes, the tension will fall to one third or one half of normal and will return to normal in from thirty to forty-five minutes. In the glaucomatous eye

the tension may remain unchanged or may fall to a lower degree. The tension returns to its former reading much more rapidly than in the normal eye and may even exceed it and not return to its original reading for one hundred minutes (P. Knapp[1]; fig. 5).

DARK ROOM TEST

According to Seidel[22] and others, this test is very useful in latent glaucoma. The patient is put in the dark room for an hour after the tension has been taken. At the end of this time the tension is again taken. In glaucomatous eyes or in eyes with latent glaucoma there is a sharp rise. If the patient then looks at a brightly lighted white wall or at the clear sky the tension falls rapidly to its former level. The

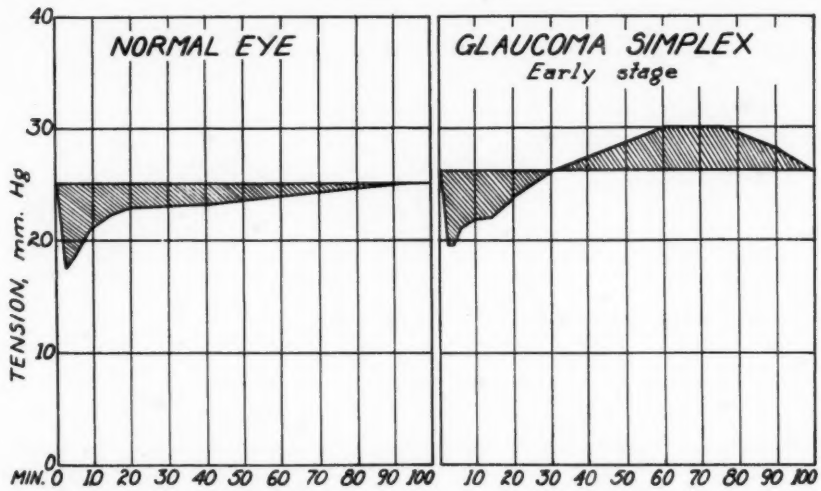

Fig. 5.—Curves for tension after massage for a normal eye and an eye in the early stage of glaucoma simplex.

variation may be from 10 to 40 mm. of mercury. In the normal eye this procedure causes no appreciable variation. This test is of particular value in the case of eyes the anterior chamber of which is shallow. The same procedure carried out in a dimly lighted room produces similar results (fig. 6).

The use of weak mydriatics will also cause the change in tension, but the sudden fall on exposure to light cannot be obtained.

YEARLY CURVE FOR SIMPLE GLAUCOMA

In simple glaucoma there is a variation in the yearly curve. If the tension is taken weekly, it is found that it is higher during the period

22. Seidel: Arch. f. Ophth. **88**:102, 1914.

from November to January. The cause is not known. Perhaps the rapid change in temperature and atmospheric pressure play a part.

VISIBLE CHANGES IN THE EYE IN EARLY SIMPLE GLAUCOMA

In early simple glaucoma no visible changes may be present. In the beginning the anterior chamber is almost always normal. A fairly shallow chamber has doubtful diagnostic value, as the shallowness varies with the refraction and is greater in hyperopic and presbyopic eyes than in normal eyes. In glaucoma in persons under 35 (so-called juvenile glaucoma) the anterior chamber often is deeper than normal.

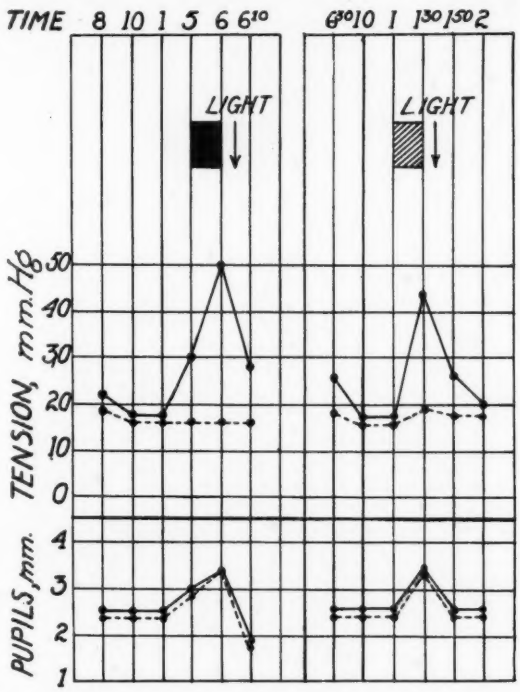

Fig. 6.—Curves for tension for a normal eye and a glaucomatous eye in the dark room test and in the test with dim light.

The size and reaction of the pupil are, as a rule, normal in the beginning. Pupillary changes, atrophy of the iris and increased congestion of the vessels of the iris make their appearance later in the disease. A. Knapp[23] reported that in many cases of early glaucoma instillation of 1 drop of a 1:1,000 solution of epinephrine hydrochloride caused pupillary dilatation, and he stated that this is an early symptom of compensated glaucoma.

23. Knapp, A.: Tr. Am. Ophth. Soc. **19**:69. 1921.

With the slit lamp the presence of excessive fine pigment dust over the iris, the posterior corneal surface and surface of the lens is noted in glaucoma. Koeppe [24] expressed the belief that this is of great significance as indicating possible latent glaucoma. As these changes cannot be differentiated from the ordinary senile changes, their presence is of doubtful diagnostic value. Often they may appear later in the disease and especially after operative intervention.

Recently a good deal of work has been done on the angle of the anterior chamber in glaucoma. There is particular interest in the presence of pigment dust and its possible obstruction of Schlemm's canal. This pigment is often present in normal eyes to some degree. Probably the amount of pigment present has a bearing on this subject. O. Barkan [25] stated that on biomicroscopic examination he has seen in many cases of glaucoma an area of blockage in the trabecula which separates Schlemm's canal from the anterior chamber, and he feels this is a causative factor.

Opacities of the lens, as Gradle [26] has pointed out, occur so frequently during the age period when glaucoma is most prevalent that their presence is of no diagnostic value and must be regarded as entirely independent of the disease. Vogt [27] pointed out that in cases of lenticular opacity in which the opacity is primarily an exfoliation of the anterior capsule, glaucoma may develop. With the slit lamp the typical picture is seen, with bluish gray clumpings at the pupillary border of the iris. These are the result of exfoliation of the anterior capsule of the lens, and possibly these clumpings may also interfere with the drainage of Schlemm's canal and in this way cause glaucoma. This is so well established that the condition has been called glaucoma capsulare. In these cases there is usually low grade glaucoma that must be watched carefully.

The typical picture of glaucomatous evacuation and atrophy does not belong to the classification of early simple glaucoma but is seen only in the later states. The diagnosis of early glaucomatous cup is difficult. If there has been a definite physiologic cup in the center of the disk, this enlarges during the early stages of increased pressure. Consequently, in beginning glaucoma there may be only a large physiologic cup present in the disk. In every case of an unusually large physiologic cup, beginning glaucoma must be kept in mind. This condition should not be confused with a true glaucomatous cup, as the normal color is present, not the greenish tinge of the glaucomatous cup.

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24. Koeppe, L.: Ber. ü. d. Versamml. d. ophth. Gesellsch. **40**:478, 1916.

25. Barkan, O.: Paper read before the American Academy of Ophthalmology and Otolaryngology, New York, Sept. 29, 1936.

26. Gradle, H., in Jackson, E.: Contributions to Ophthalmic Science, Menasha, Wis., George Banta Publishing Company, 1926, p. 255.

27. Vogt, A.: Schweiz. med. Wchnschr. **56**:413, 1926.

Also, the halo is absent, and there always remains at least a narrow band of normal nerve tissue. In cases in which a large physiologic cup is present careful sketches should be made so that one can determine from time to time whether it has increased in size. In cases in which no physiologic cup is present the earliest signs of excavation are a very flat shallow cupping beginning sharply from both the nasal and the temporal side. The vessels in this instance show only a very slight dip at the margin of the disk.

The time required to produce a glaucomatous cup varies greatly, depending on the degree of tension and the structure of the eye. There are cases in which the disk withstands rather high tension for a long time without signs of cupping, and, conversely, there are eyes in which the tension is relatively low and in which a glaucomatous cup develops early.

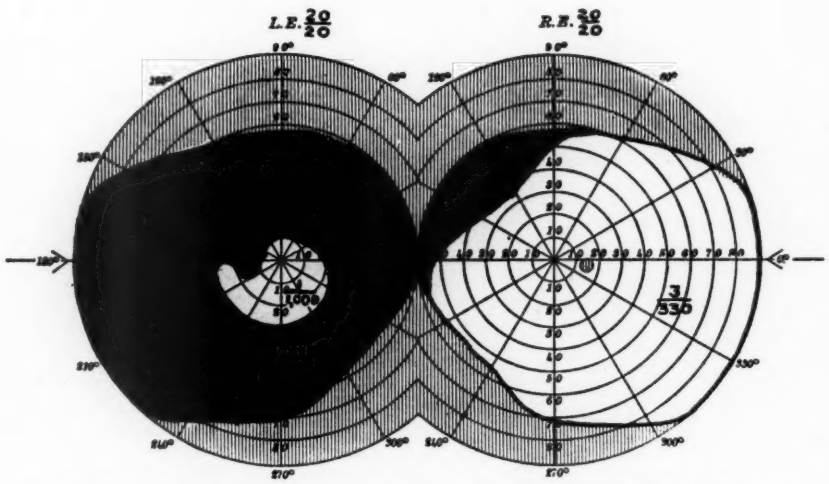

Fig. 7.—Visual fields at the beginning of primary simple glaucoma. The left eye shows an early cut central isopter as determined by the screen study; the right eye, an early cut nasal field, as determined by the perimeter.

CHANGES IN THE VISUAL FIELD

In primary simple glaucoma the disturbance of function is primarily the result of pressure on the optic nerve and its subsequent excavation. This atrophy of the nerve has the peculiar tendency to be involved by bundles, thus producing characteristic defects in the field.

At the beginning of the disease and even in the presence of a definite partial atrophy and excavation the peripheral field is usually normal except for a contraction of the nasal field, which may appear early in the disease. The contraction is not great and does not spread uniformly over the entire nasal field (fig. 7).

Very early in the disease scotomas extending from the blindspot are present, the form of which follows the course of the bundles of nerve fibers in the retina. Seidel [22] found that in very early cases the so-called Seidel scotoma often was the only indication of the disease. This consists of a tuftlike or flamelike appendage to the blindspot or, when the condition is more advanced, of crescentic or sickle-shaped, narrow, pointed defects arching from one or both poles of the blindspot around the fixation area (fig. 8). These may be inconstant and vary from day to day or may develop into the typical Bjerrum scotoma. This consists of a semilunar scotoma extending from the blindspot above or below, or above and below (fig. 8). In the latter form the defects may meet in the nasal half of the field and produce a ring scotoma. The bundles of nerve fibers course about the fixation area in

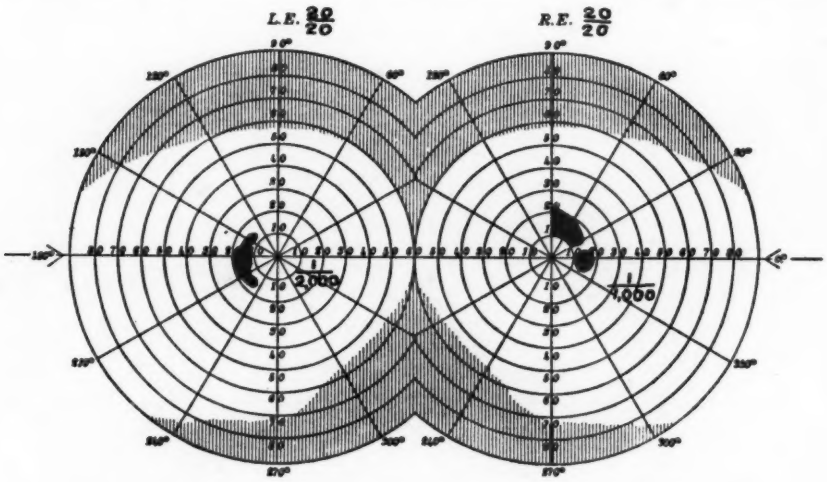

Fig. 8.—Visual fields in the early stage of primary simple glaucoma. The left field shows Seidel's scotoma; the right field, Bjerrum's scotoma.

the temporal half of the retina and end in the median raphe. As more damage is done to these fibers one observes a sharply defined defect in the nasal quadrant, the so-called nasal step of Rönne [28] (fig. 9). This is more frequently found in the lower quadrant or in both the lower and the upper quadrant, in the latter case producing a complete defect of the nasal field. This defect of the nasal field almost always arises from the center rather than from the periphery of the field. The contraction of the peripheral field usually occurs only in the later stages of the disease. At times the only change in the central field consists of definite enlargement of the blindspot without a defect in the peripheral field.

28. Rönne, H.: Arch. f. Ophth. **71**:52, 1909.

From this it is apparent that the perimeter does not suffice in the study of early stages of glaucoma, and careful study on the screen is necessary in order to detect the earliest signs of glaucomatous changes in the field.

Central vision remains to the last, so that vision as determined by tests with Snellen's letters is no indication of the progress of the disease.

CHANGES OF REFRACTION

The most significant change in refraction in glaucoma is a rather rapid loss of accommodation, requiring frequent changes in reading glasses. Mild myopia may also occur, owing to forward displacement of the lens with decrease in the depth of the anterior chamber. A rapid change in presbyopia should always make one suspicious of glaucoma.

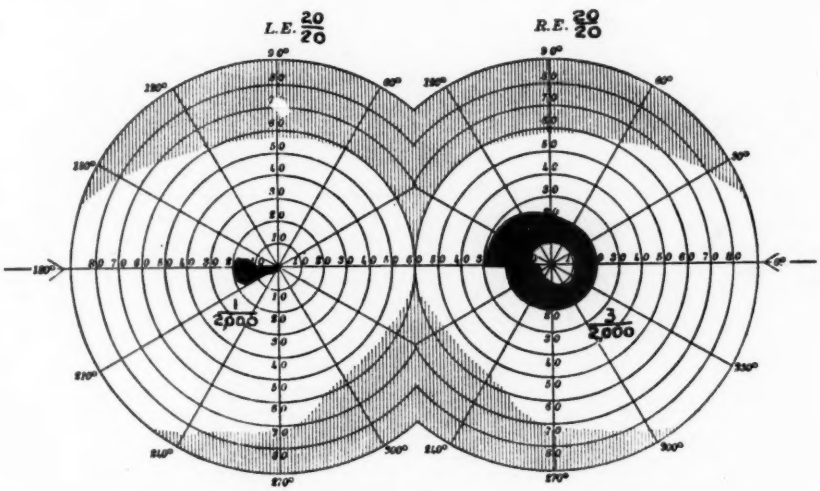

Fig. 9.—Visual fields showing later damage. The left field shows an atypical nerve fiber bundle defect; the right field, a double arcuate scotoma with Rönne's nasal step.

COURSE OF THE DISEASE

Chronic simple glaucoma begins without symptoms such as pain or visual disturbance, so that frequently the disease is well advanced when the ophthalmologist is consulted. If the disease is untreated the well known picture of glaucomatous cupping, atrophy and blindness ensues. There is also a tendency for glaucoma simplex to develop into the chronic inflammatory type of glaucoma. In unilateral glaucoma the apparently healthy eye must be watched carefully, as in the majority of cases both eyes are involved.

DIAGNOSIS OF SIMPLE GLAUCOMA

The presence of increased intra-ocular tension, excavation of the nerve head and atrophy and disturbances of function is sufficient to warrant a diagnosis of glaucoma. Until the aforementioned signs have been ruled out glaucoma cannot be excluded. The tension must be taken with a tonometer, as palpation is too unreliable.

In the early stages diagnosis is sometimes difficult. If increased pressure is present and there are scotomas of the type described, the diagnosis is certain, even though the nerve may show no visible change. The presence of normal tonometer readings in the middle of the afternoon does not exclude glaucoma in cases in which there is a scotoma associated with the blindspot, especially with an accompanying suspicious early change in the disk. Glaucoma cannot be ruled out until the twenty-four hour curve for tension has been recorded over a period of two or three days.

INCIDENCE

Primary glaucoma is a disease of old age, with a sharp rise in the curve for tension after 45 years of age, and is more frequent in women than in men. It is rather common among the Jewish race and is supposedly more frequent in the Orient than in Europe. It is also more frequent in the Negro race than in the white race. Its hereditary tendency has long been recognized. Both eyes are involved in most instances, and the condition has a tendency to run a similar course in the two eyes. It is often associated with arteriosclerosis and cardiovascular changes, which are probably coincident and are to be considered principally as additional signs of old age. The disease is more prevalent in hyperopes than in myopes.

MANAGEMENT OF SIMPLE GLAUCOMA

The treatment of simple glaucoma can be divided into two general headings: (1) medical and (2) surgical.

Medical Management.—The treatment discussed here is limited to that for early simple glaucoma.

In the medical care of glaucoma both the oculist and the patient assume a great responsibility. It is necessary to impress on the patient the seriousness of the disease and the necessity of constant medication and periodic observation. In the periodic examination, tension, examination of the fundus and studies of the field are important. Of these, the most important is the careful detailed screen study of the defect of the field. The changes in the field are the most important indication as to the progress of the disease, as some eyes can tolerate a tension slightly higher than normal without change while in some an apparently normal tension is sufficient to produce changes.

Miotics—Pilocarpine and Physostigmine: In spite of recently advocated medication, pilocarpine and physostigmine still hold the first place in importance. Their action is to stimulate the parasympathetic end-organs of the oculomotor nerve, producing contraction of the pupil. The most commonly accepted theory of their action is that the base of the iris is freed and the angle of the anterior chamber made more open, thus opening Schlemm's canal, and this increases the absorbing surface of the iris.

Pilocarpine hydrochloride or pilocarpine nitrate is given in a 0.5 to a 2 per cent solution, depending on the action desired. In some patients who are sensitive a 0.5 per cent solution will keep the pupil contracted for from eight to twelve hours. With the average patient a 1 or a 2 per cent solution of pilocarpine given every four to six hours controls the tension during the day. It is advisable to take the tension at the end of the four to six hour period to determine whether or not it has risen above the normal, in other words, to determine whether the drops maintain the normal tension for that period of time. On the basis of the knowledge that the tension is apt to rise during the night, a drop must be instilled just before retiring. In patients for whom the curve for the tension shows a definite rise at night, an ointment containing from 1 to 2 per cent of pilocarpine applied at night just before retiring is indicated. In the group in whom the tension is normal during the day but rises at night, a 1 to 2 per cent solution of pilocarpine instilled at night just before retiring is often sufficient. In some instances a pilocarpine ointment at night controls the curve for tension efficiently. In these cases, particularly, careful periodic examination of the central fields is important.

Physostigmine salycilate or physostigmine sulfate can be employed in a 0.2 to a 0.5 per cent solution. The action of physostigmine is stronger than that of pilocarpine, but it has the disadvantage of being slightly irritating and in most patients causes a dermatitis venenata in a relatively short time. It is pretty generally accepted that long-continued physostigmine therapy is detrimental to the eye, so that any case in which the use of physostigmine is required for the maintenance of normal tension must be considered a surgical one.

Pilocarpine also, after being used over a long period, is prone to produce a dermatitis with accompanying conjunctivitis. When this occurs the patient is treated with solution of physostigmine for three or four days, during which time the dermatitis and conjunctivitis disappear. It is a good rule to have the patient use solution of physostigmine as a routine for two or three days of every month.

In cases in which miotics do not control the tension, surgical intervention, as a rule, is indicated. In such cases the sooner surgical

intervention is advised the better. Certain circumstances, however, may make a delay advisable or operation impossible, and under these conditions some of the more recently advocated therapeutic measures may be indicated. Some of these conditions are: a very old and feeble patient, refusal of operation, a one-eyed person who refuses operation.

Epinephrine: Of the newer therapeutic agents, epinephrine and its derivatives are most useful. Wessely [29] was the first to show experimentally the effect of epinephrine on the intra-ocular tension. He found that both simple instillation and subconjunctival injection of epinephrine hydrochloride reduced the tension in the eyes of animals. Ordinary epinephrine hydrochloride instilled is effective in only a small percentage of cases, but subconjunctival injection is followed by a fall in the tension in most cases that reaches its maximum from twelve to fourteen hours after the injection, the dose being 4 minims (0.24 cc.) of a 1:1,000 solution of epinephrine hydrochloride injected under the conjunctiva at the temporal side of the globe. This causes a rise in blood pressure, which in some patients is accompanied by systemic symptoms.

I have found the method of Gradle [30] effective a good many times. This produces almost as much effect as an injection but without systemic reactions. The conjunctival sac is anesthetized, and from 4 to 5 minims (0.24 to 0.3 cc.) of a 1:1,000 solution of epinephrine hydrochloride is dropped on a small cotton pledget, placed in the upper cul-de-sac and left for two and one-half minutes.

Hamburger [31] advised synthetic dextrorotatory epinephrine, which he called *Glaukosan*, for the injection. To avoid injection he used a 2 per cent solution of epinephrine hydrochloride, to which he added an optically inactive substance produced in the manufacture of epinephrine, which he called *Links-Glaukosan* or *Laevo-Glaukosan*. Two drops of the solution are placed in the inner angle, and the lids are held open for thirty seconds. This is repeated from three to five times. More recently suprarenin bitartrate in a 2 per cent solution has been used in this country, and within the past few months I have had occasion to use a new 1:100 solution of epinephrine hydrochloride, which is more stable than any of the synthetic epinephrine products.

All these methods apparently have practically the same effect on the tension. In most cases they produce a fall of the tension below 25 mm. (Schiötz). It is necessary to use miotics after the treatment in the usual manner. The use of epinephrine in conjunction with miotics may keep the tension normal for from a week to as long as six weeks. Often

29. Wessely, K.: Ber. ü. d. Versamml. d. ophth. Gesellsch. **28**:69, 1901.

30. Gradle, H.: The Use of Epinephrine in Ocular Hypertension, J. A. M. A. **84**:675 (Feb. 28) 1925.

31. Hamburger, C.: Klin. Monatsbl. f. Augenh. **76**:400, 1926.

later treatments are not as effective as the first treatment, and there is a marked variation in response to the treatment. If the first treatment is ineffective, further attempts are useless.

The possibility of acute glaucoma resulting from the use of these epinephrine preparations must be kept in mind, even though this is infrequent in simple glaucoma. The use of physostigmine before the treatment and every twenty minutes for the first hour after the administration of epinephrine seems to help prevent this. In my experience the use of epinephrine wicks seems less likely to produce this complication. According to Gifford,[32] the possibility of an acute attack speaks against the use of epinephrine ointment at home.

Other Drugs.—Ergotamine tartrate (gynergen), a derivative of ergot, supposedly depresses the end-organs of the sympathetic nervous system and thus should help glaucoma by decreasing the permeability of the vessels. The dose is one-half ampule for injection or 1/60 grain (0.001 Gm.) by mouth. Injections are given twice daily, or the oral dose is given two or three times a day. This treatment appears to help in some cases but is very expensive and, in my opinion, does not offer sufficient advantage over other methods to warrant its use.

Recently extract of adrenal cortex has received a good deal of publicity. My experience with its use in a relatively few cases has been that it has no effect on the intra-ocular tension in glaucomatous eyes. There have been several reports in the literature confirming this experience. Woods [33] found adrenal cortex extract ineffective in glaucoma, whether given intramuscularly or intravenously.

The effect of any of the aforementioned therapeutic agents, aside from miotics, is a temporary one, and their value in simple glaucoma is chiefly in cases in which operation must be delayed.

GENERAL CARE

The patient should have a careful physical examination, and measures should be taken to control the blood pressure and obvious pathologic conditions. The so-called vasoneurotic diathesis (unstable vasomotor mechanism, i. e., tachycardia, angioneurotic edema and urticaria) and the related bronchial asthma seem to have a relationship to glaucoma. All frank foci of infection should be cared for. One is not justified, however, in recommending radical procedures, such as the removal of tonsils suspected to be infected. The patient should also be warned against physical and mental excitation. The average use of the eyes for near work can also be allowed.

32. Gifford, S. F.: Am. J. Ophth. **11**:628, 1928.

33. Woods, A. C.: The Use of an Extract of Adrenal Cortex in Glaucoma, Arch. Ophth. **14**:936 (Dec.) 1935.

Surgical Management.—In cases in which miotics do not control the tension, surgical intervention is indicated. The most important indication of the progress of the disease is changes in the field as determined by a careful, detailed screen study. Any increase in the size of the scotomas is an indication for operation when the tension has not fallen to well within the normal limits.

Cyclodialysis is the operation of choice for early simple glaucoma when the tension is not above 40 mm. (Schiötz). If properly done it opens up a space between the sclera and the choroid for the absorption of aqueous. The severing of the ciliary nerves and vessels over the area of the operation also reduces the formation of the aqueous. I have found the operation successful in a high percentage of properly selected cases. Meller [34] stated that he obtained successful results in about 70 per cent of his cases. In cases in which the procedure has not been successful it can be repeated two or three times in different quadrants. In performing the operation certain details have aided in increasing the percentage of successful results. The patient is given a preparation of calcium phosphate and calcium lactate for three days preoperatively to reduce the bleeding to a minimum, the dose being a teaspoonful in a glass of water three times a day before meals. The scleral incision is made at a measured distance of from 4 to 4.5 mm. from the limbus. In making the incision the keratome is tilted away from the limbus; this makes possible an oblique incision through which the spatula is easily introduced into the anterior chamber. The tip of the spatula should be introduced almost to the pupillary area so that separation is obtained over a large area. The operation is not difficult, and should it fail it in no way interferes with any of the filtering operations. In addition, it does not have the disadvantage of the filtering operations, such as the production of sudden drops of tension, with resultant retinal hemorrhage or the possibility of late infection. On rare occasions there is hypotony with associated lenticular changes. These conditions, however, occur in so few cases that they can be practically disregarded. When the operation is successful the intra-ocular tension is reduced to normal; miotics, however, must be continued to assure continued control of the tension.

In cases in which cyclodialysis is unsuccessful one of the filtering operations must be resorted to, the type of the operation depending primarily on the choice of the operator.

It is possible that in the future the goniotomy operation of O. Barkan may prove to be the proper procedure in glaucoma simplex.

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34. Meller, J.: Personal communication to the author, 1935.

PROGNOSIS

In early glaucoma simplex the prognosis is rather good, and in many patients vision can be preserved throughout life by miotics controlled by careful periodic observation. Even in the early stages of the non-malignant type for which surgical intervention is required, the outlook for retention of vision is good. In all cases, however, continued periodic observation is necessary. The most important indication of the progress of the disease is the changes in the field of vision, particularly changes in the central field, as noted in studies on the screen.

Obituary

WARD ANDREWS HOLDEN, M.D.
1866-1937

Ward Andrews Holden died suddenly on January 24, at the University Club, New York, in his seventy-third year. His death removes another outstanding figure from American ophthalmology.

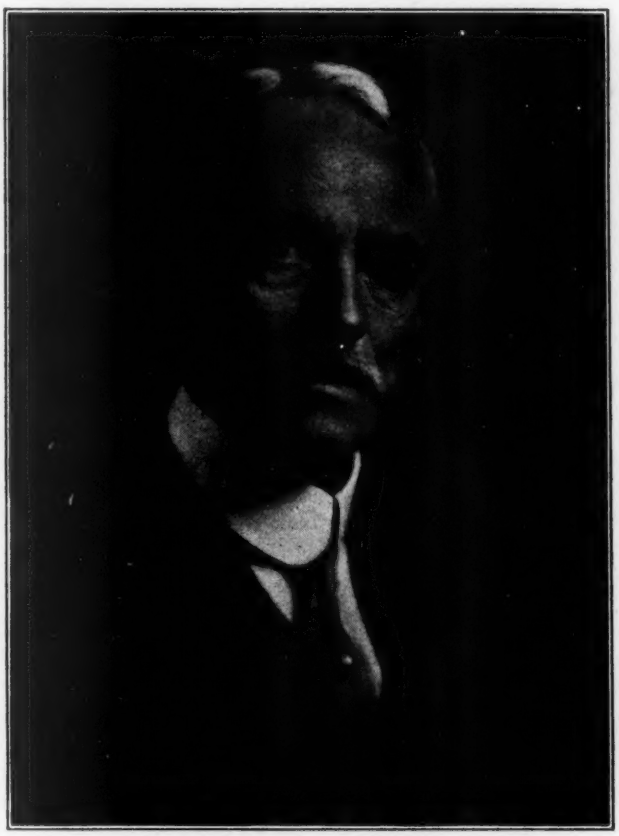
He was born in Marietta, Ohio, the son of William and Sarah Andrews Holden, on Feb. 15, 1866. He received the degree of Bachelor of Arts in 1884 from Marietta College, of which his grandfather, Israel Ward Andrews, was president; and the degree of Doctor of Medicine from the Medical College of Ohio, Cincinnati, in 1887. After taking postgraduate work at the University of Göttingen, the University of Vienna and the University of Marburg, from 1888 to 1890, he settled in New York and became associated with Dr. Herman Knapp in his private office. He was connected with the New York Ophthalmic and Aural Institute, and the Vanderbilt Clinic of the College of Physicians and Surgeons. From the first Holden gave much attention to work in the laboratory, where he gave courses for a number of years on the embryology and pathology of the eye; he wrote short textbooks on each of these subjects in 1893. At the Pathological Institute of the New York State Hospital he made his important investigations on toxic amblyopias, in which he demonstrated the degeneration of the retinal ganglion cells, findings which were later verified by Birch-Hirschfeld. This work was published under the following headings: "Experimental Quinine Amblyopia" and "Pathology of Amblyopia Following Profuse Hemorrhage and of That Following the Ingestion of Methyl Alcohol, with Remarks on the Pathogenesis of Optic Nerve Atrophy in General."[1] In 1898 and 1899 he published two articles, "A Case of Excessive Distortion of the Optic Chiasm in Acromegalia" and "The Sequence of Changes in the Optic Chiasm Produced by Acromegalia as Exemplified in Three Cases."[2] These were among the earliest publications on the important relation of the eye to the pituitary region.

1. Holden, W. A.: Experimental Quinine Amblyopia, Arch. Ophth. **27**:457, 1899; Pathology of Amblyopia Following Profuse Hemorrhage and of That Following the Ingestion of Methyl Alcohol, with Remarks on the Pathogenesis of Optic-Nerve Atrophy in General, ibid. **28**:125, 1899.

2. Holden, W. A.: A Case of Excessive Distortion of the Optic Chiasm in Acromegalia, Arch. Neurol. & Psychopath. **1**:699, 1898; The Sequence of Changes in the Optic Chiasm Produced by Acromegalia, as Exemplified in Three Cases, ibid. **2**:575, 1899.

He then became interested in the association of the eyes with nervous diseases, and served as attending ophthalmologist to the Neurological Institute from its beginning on Sixty-Seventh Street, where he worked faithfully until the institute moved up to its new quarters.

In 1904 Holden became chief of clinic in the ophthalmic department of the Vanderbilt Clinic and honorary surgeon to the Herman Knapp Memorial Eye Hospital.



WARD ANDREWS HOLDEN, M.D.
1866-1937

In 1918 he wrote a short paper, "The Eye in Nervous Disease: An Introduction to Neurological Ophthalmology, prepared for the New York Neurological School for Army Medical Officers." [3]

3. Holden, W. A.: The Eye in Nervous Disease: An Introduction to Neurological Ophthalmology, Prepared for the New York Neurological School for Army Medical Officers, Arch. Ophth. **47**:221, 1918.

As one of the editors of the *Archives of Ophthalmology* from 1893 to 1928, he did yeoman service in the cause of scientific ophthalmology in this country.

Holden was of a quiet and retiring nature. He had marked ability and many interests of an artistic nature outside of his profession which made an impress on his medical thought and action. He had an unusually alert mind, and his literary style was lucid. He never took an active part in medical meetings but succeeded in making real contributions to ophthalmology, for which his name will always be remembered. He was fond of music and art and had marked ability in drawing. This talent proved of great help in his profession and later furnished activity during many summer holidays, which usually resulted in a series of delightful pastel sketches.

Holden was one of the original members of the Charaka Club, a club devoted to the historical, literary and artistic sides of medicine. He never married, and for many years the University Club was his second home; it seemed fitting that he should have been listening there to a symphony concert on the radio when the fatal stroke of paralysis came. He left no near relative except a sister, Mrs. Elizabeth Holden Canda, of Pomfret, Conn.

ARNOLD KNAPP.

* * * * * *

When I returned from Germany in the early nineties, whither I had gone to study neurology, I realized that in the diagnosis of nervous diseases the use of the ophthalmoscope was essential. Hence I offered myself as voluntary assistant in the New York Ophthalmic and Aural Institute, which was under the direction of the leading ophthalmologist of the country, Dr. Hermann Knapp. I soon found there a young man who was earnest, efficient and affable. I stuck to him as a burr does to the bushy tail of a dog. From those days dated a friendship that existed nearly half a century. I had not been in the outpatient department of the hospital long before he began to give me instruction. After the work of the afternoon was finished we would walk uptown. The hospital was at Twelfth Street, and we both lived in Thirty-Eighth Street. On the way we were likely to stop at one of the book shops which were then in Union Square. I soon found that he knew not only his mother tongue but German, Italian and French, and as I, even in those early days, was interested in letters, we had many interesting discussions. I use the word discussion, but imparting of information would be a more correct term. He made no parade of his knowledge of literature, but he was ready to reveal it on request. Many of our evenings in the winter we spent in the gallery of the Metropolitan Opera House or listened to the New York Philharmonic Symphony or other symphony societies. He had the same sensitiveness for music

that he had for literature and art, and by art I mean painting. All that I know of pictures he taught me. Even in our later life he would take me from gallery to exhibition and point out the merits or the demerits of the exhibits. He was himself a water colorist of high order, and it was a pleasure to observe him showing his pictures so modestly to the members of the Charaka Club. When Joseph Frankel and I established the Neurological Institute, Dr. Holden became the attending ophthalmologist, and he worked hard, faithfully and efficiently to our great advantage for nearly fifteen years.

Dr. Holden was what the French call a savant, that is, a person who knows all there is to be known about the subject of his profession and in addition knows something about nearly everything else. His outstanding characteristics were courtesy, decorum, unselfishness and modesty. Therefore, he was a gentleman; in fact, of all the gentlemen I have known, Ward A. Holden tops the list.

JOSEPH COLLINS.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Biochemistry

RETINAL METABOLISM. R. CAMPOS, Ann. di ottal. e clin. ocul. **64:** 457 (July); 538 (Aug.); 577 (Sept.) 1936.

The work reported, which received the Cirincione prize for 1935, included studies of the respiration of the retina in the rat, the rabbit and a few human beings. The Warburg apparatus was employed for both the direct and the indirect methods. The literature on previous work on the retina by Warburg and others is reviewed.

In the work with the rat and the rabbit a marked difference was found between the respiration estimated by the direct method and that estimated by the indirect method. The high figures obtained by the indirect method represent something peculiar to the retina, as compared with other tissues, which is apparently the result of glycolysis. When the retina was first dark-adapted, the consumption of oxygen was much increased, as compared with that in the light-adapted retina. These findings were confirmed in the monkey, and Campos considers this as proof of Ovio's theory that adaptation is an expression of the retinal metabolism.

The gas exchange of the human retina was examined by both the direct and the indirect method. The material was eyes removed from six to ten days after a penetrating wound, one normal eye and three eyes with secondary glaucoma. There was little change shown in the results following wounds for periods up to ten days. The retina in all these eyes showed an active respiration, with marked aerobic and anaerobic glycolysis, the former being more marked than the latter. There was no excess of fermentation, and the Pasteur reaction was low or negative. The findings agreed with those found in normal rabbits' eyes. In eyes with further changes in the retina such as occurred from twenty-four hours to ten days after injury, aerobic glycolysis was reduced, while anaerobic glycolysis remained nearly normal.

The macular region was compared with the peripheral part of the retina in its respiration. The peripheral part of the retina showed a more active respiration in the relatively normal eye, while in an eye with absolute glaucoma this condition was reversed.

The choroid and retina of pigmented and albino rats were investigated by the Warburg method. The choroid of albino animals showed the more active respiration. The retina of the albino rats showed a slightly more active respiration, while its aerobic glycolysis was definitely less than that of pigmented animals. A bibliography is included.

S. R. GIFFORD.

Blind

BLINDNESS IN WEST CHINA. E. R. CUNNINGHAM, Chinese M. J. **50:** 1507 (Oct.) 1936.

In this paper brief mention is made of reports and statements relating to blindness, published by ophthalmologists working in China. A list is given of the causes of blindness found in 749 blind eyes in 506 patients. It is pointed out that the acute ophthalmias which are so closely associated with corneal ulceration are undoubtedly at the bottom of most of the blindness. Trachoma also plays a large part and is often allied with acute conjunctivitis. When economic conditions are disturbed, keratomalacia may produce much blindness.

Blindness caused by congenital or hereditary disorders was met with only rarely.

S. H. MCKEE.

Conjunctiva

A RETENTION CYST OF UNUSUAL SIZE, PROBABLY OF KRAUSE'S GLAND, SIMULATING ANGIOMA OF THE ORBIT. D. V. GIRI, Brit. J. Ophth. **20:** 621 (Nov.) 1936.

A cystic tumor which was smooth, mobile and nonadhesive to the skin, situated at the upper inner angle of the orbit, was seen in a girl of 9 years. On removal it proved to be a white translucent oval cyst about 3 cm. long and 1.7 cm. broad. Histologically the cyst was lined with two layers of cells, a deeper layer of flattened and a more superficial layer of cubical cells. In parts there were several layers of cells resembling those of altered conjunctiva. There was a well marked wall of fibrous tissue, and here and there were nodules of lymphocytes. The diagnosis was a probable retention cyst arising from the conjunctiva or a gland of Krause. The growth was not an ordinary dermoid.

The article is illustrated.

W. ZENTMAYER.

HISTOLOGIC EXAMINATION OF PROWAZEK'S BODIES AND STATISTICS ON THEIR GEOGRAPHIC DISTRIBUTION. Y. ONISI, Klin. Monatsbl. f. Augenh. **96:** 797 (June) 1936.

In the first chapter of this paper the histologic technic of the preparation of Prowazek's bodies is given, and their relation to trachoma and to inclusion blennorrhea is discussed. The second chapter contains elaborate statistical data and tables on the geographic distribution of Prowazek's bodies in the northern part of Japan. Smears and sections in 1,448 cases of trachoma were examined for a period of five years. Prowazek's bodies were found in 54 cases, or 3.73 per cent. This percentage was lower than in the southern districts of Japan. Onisi therefore concludes that the southern climate is more favorable for the development of Prowazek's bodies. In most of the cases in which these bodies were present severe papillary hyperplasia of the conjunctiva was found clinically.

K. L. STOLL.

Cornea and Sclera

THE PATHOLOGY OF LATTICE AND NODULAR DYSTROPHY OF THE CORNEA. F. H. MAURY, Am. J. Ophth. **19:** 866 (Oct.) 1936.

Maury reports a case of bilateral corneal dystrophy in a 58 year old man one of whose eyes was phthisical, apparently as a result of surgical intervention. As this eye did not have useful vision and was persistently painful, it was enucleated and examined. Maury gives the histologic findings and reviews the literature from the standpoint of the pathologic reports. He gives the following summary:

"The clinical and histological findings in a case of combined lattice and nodular dystrophy of the cornea are reported. The lattice and nodular lesions both showed the same histological features. The primary lesion is a hyalin-lipoid degeneration of localized portions of the corneal lamellae."

W. S. REESE.

GUMMA OF THE CORNEA. A. VOLKOVA, Sovet. vestnik oftal. **9:** 350, 1936.

The cases of two young female patients with gumma of the cornea are presented because of the rarity of the condition. The following were the outstanding features: parenchymatous keratitis and a yellow-gray tumor with sharply demarcated borders at the site of the densest opacity of the cornea. The tumor gradually became vascularized and elevated 1 mm. above the surface of the cornea. In the first patient it had the shape of a mushroom cap; in the second patient the shape was oval. In the first patient the Wassermann reaction of the blood was negative at first and became positive after a provocative injection of neoarsphenamine. The gumma absorbed after six injections of neoarsphenamine. In the second patient the gumma underwent partial necrosis associated with marked hypotony, because of the fistulization of the cornea. In both patients a milky-white scar formed at the site of the gumma after a period of several months of antisyphilitic treatment.

O. SITCHEVSKA.

CONTRIBUTION TO THE KNOWLEDGE OF THE MARGINAL CORNEAL ULCER. P. S. SOUDAKOFF, Chinese M. J. **50:** 1393 (Oct.) 1936.

Five cases of ring-shaped marginal corneal ulcer are described. In one case the condition was diagnosed as an endogenous infection and in the other four cases as an exogenous infection. In three cases of the latter group pathogenic micro-organisms were isolated, while in one case the causative micro-organism was not established.

The ring-shaped marginal ulcer usually has a moderately severe course, with tendency to recurrence. In exceptional cases, as in the four reported in this article, the ulcer was progressive and the outcome was a complete leukoma of the cornea.

The clinical facts indicate that the cornea that has once been affected by a ring-shaped ulcer is predisposed to recurrence at the inner border of the previously formed scar. There is a considerable bibliography.

S. H. MCKEE.

Experimental Pathology

PRECIPITINS IN THE OCULAR TISSUES OF RABBITS GENERALLY AND LOCALLY IMMUNIZED WITH CRYSTALLINE EGG ALBUMIN. R. THOMPSON, E. GALLARDO and D. KHORAZO, Am. J. Ophth. **19:** 852 (Oct.) 1936.

This article does not lend itself to abstracting. Five tables are given showing the results of experiments on albino rabbits. The animals were immunized intravenously or given single or repeated local injections of crystallized egg albumin into the anterior chamber or into the cornea. The authors remarked the presence, following the local injection of antigen, of precipitins in the corneal tissue earlier and in greater concentration than elsewhere. Their finding of the absence of precipitins in the aqueous until the serum contained them and until local inflammation had occurred corroborates the results noted in previous work. These experiments indicate that the aqueous does not reflect the state of the surrounding tissues.

W. S. REESE.

OCULO CARDIAC REFLEX AS MODIFIED BY INDUCED HYPERTHERMIA. P. VALLERY-RADOT, G. MAURIC and J. LEMANT, Compt. rend. Soc. de biol. **123:** 670, 1936.

In a series of subjects hyperthermia was induced by intramuscular injections of a 1 per cent solution of sulfur in oil, and the cardiac reaction to ocular compression was studied. Under average normal conditions the pulse rate is reduced four to twelve beats, but in the cases of hyperthermia the oculocardiac reflex became attenuated, obliterated or reversed. The authors conclude that hyperthermia provokes a tendency to sympathicotonia, which may persist for from three to six days after the curve for temperature has become level.

J. E. LEBENSOHN.

General Diseases

PERNICIOUS ANEMIA WITH RETROBULBAR NEURITIS. G. TALBOT, Brit. J. Ophth. **20:** 619 (Nov.) 1936.

A man aged 55 years had pernicious anemia, which had been recognized and treated over a period of seven years. Vision had been failing for one year in each eye and equaled counting fingers. The optic nerve did not show atrophy, but one disk was a little paler than the other. The visual fields were full but showed a central relative scotoma. The pupils reacted to light.

Talbot suggests that the ocular condition might be accounted for by a demyelinization of the optic nerve comparable to the demyelinization of the posterolateral columns found in subacute combined degeneration. The comparatively slight interference with the axis-cylinders would account for the nondevelopment of optic atrophy.

W. ZENTMAYER.

SECONDARY ZONULAR OPACITY OF THE CORNEA IN POLYARTHRITIS CHRONICA LEUKOCYTOTICA (STILL) AND LYMPHOCYTOTICA (RHONHEIMER). J. KARSCH, Arch. f. Augenh. **110:** 106, 1936.

Polyarthritis chronica leukocytotica, or Still's disease, is an infectious disease of childhood of unknown etiology. The signs are fever,

enlargement of the spleen and the lymph glands and an inflammation of the small joints in the form of periarthritis. The bones and cartilages often remain normal for years. The fluid in the cavities of the joints contains polymorphonuclear leukocytes. The disease usually begins at about the age of 8 and may last for years. Adhesive pericarditis frequently develops.

The form described by Rhonheimer differs but little from this form, with the exception that the fluid aspirated from the joints contains lymphocytes.

Karsch reports two cases of this disease in both of which there was an old iridocyclitis and zonular opacity of the cornea. Syphilis and tuberculosis could be excluded from the diagnosis. He considers the involvement of the cornea and iris as part of the picture of the disease.

F. H. ADLER.

Glaucoma

CYCLODIALYSIS IN THE TREATMENT OF CHRONIC GLAUCOMA. BAILLIART and LAIGNIER, Bull. Soc. d'opht. de Paris, October 1935, p. 636.

Cyclodialysis, which is used in Poland, Germany, Austria, Hungary and Czechoslovakia in the treatment of chronic glaucoma, is rarely the technic in France, because of the influence of Lagrange and his method of sclerotomy. From their use of cyclodialysis and from the discussion of the procedure as found in the literature, Bailliart and Laignier conclude that it is a simple operation, inoffensive and, without doubt, efficacious. Its action is not as lasting as sclerotomy, but it may be repeated, with little danger.

L. L. MAYER.

NEWER KNOWLEDGE OF THE DIAGNOSIS AND THERAPY OF PRIMARY GLAUCOMA. A. BRÜCKNER, Schweiz. med. Wchnschr. **66**: 1264 (Dec. 12) 1936.

Brückner emphasizes the hereditary nature of glaucoma, showing how it may attack successive generations in the same decade of life. The theory of edema with changes in the p_H of the intra-ocular fluids as observed by Fisher and by Meesmann is considered. The influence of the vegetative nervous system and of climatic conditions is alluded to. Early subjective symptoms of the insidious chronic glaucoma may be a feeling of fulness in the eye or headache. Various tests to be made in the preglaucomatous stage are outlined, and the importance of the taking of tension periodically during the twenty-four hours of the day is emphasized. In the treatment the usual miotics at times control tension. A choline derivative which affects the parasympathetic nervous system has been of great success in Brückner's hands. Various operative procedures are discussed, and the indications for each type are outlined. The injection of alcohol relieves the pain in cases of absolute glaucoma.

L. L. MAYER.

Hygiene, Sociology, Education and History

A New Eye Shield for Use in Industry. C. G. Kay Sharp, Brit. J. Ophth. **20:** 683 (Dec.) 1936.

The factors which a miner's eye shield must embody are: no diminution of illumination; free ventilation in front of the face, in use or out of use, without removal; strength; light weight, and ease with which the apparatus can be put on and taken off. Sharp has devised a shield embodying these factors. It consists of a fiber, flexible head-band with a sliding adjustment to fit any head. On the front of the head-band is a metal plate to which is fitted a metal projection carrying a double ball and socket joint. At the distal end of this joint is an oblong metal frame carrying laminated glass, in the case of the miner's protector, and mica or gauze in other trades.

W. Zentmayer.

Injuries

Notes on an Eyelash Carried by a Perforating Injury into the Posterior Aqueous Chamber and Removed Eleven Weeks Later. L. H. Savin, Brit. J. Ophth. **20:** 609 (Nov.) 1936.

Following a perforating wound in the upper nasal quadrant of the cornea the iris became attached to the whole posterior length of the wound. About seven weeks later an attempt was made to divide the anterior synechiae with de Wecker's scissors. Later it was seen that a fine strand remained. With a Ziegler knife needle an attempt was made to divide the strand, but as it was not taut it was pulled on by the knife, and an eyelash was drawn down from behind the iris. The lash was subsequently removed through a keratome incision. The eye recovered, with vision of 6/9.

W. Zentmayer.

Industrial Eye Injuries and Their Prevention. J. Minton, Brit. J. Ophth. **20:** 673 (Dec.) 1936.

This paper is based on a study of patients who were seen in the casualty department of the Royal Eye Hospital, London. In 1935, 10,786 patients were seen in the casualty department, of whom approximately 6,500 were suffering from industrial injuries of the eye. Foreign bodies in the cornea formed the bulk of these injuries. Approximately 95 per cent of these patients were occupied in metal and engineering trades. Foreign bodies in the subtarsal region were rare and were seen only in those occupied in loading and transport trades. Corneal abrasions formed the next largest group of injuries. Twenty-seven patients had intra-ocular foreign bodies. Four eyes had to be excised; traumatic cataract developed in 13. Ten patients were discharged with vision of 6/12 or better. Twenty-two cases of perforating injury to the eye were observed. Seven eyes had to be excised; 15 patients were discharged with a vision of 6/18 or better. Chemical burns were comparatively rare, probably because there are few chemical factories in the vicinity of the hospital. Injuries from lime, concrete and cement were about as common as perforating injuries. Conjunctivitis in persons engaged in electric welding was uncommon. In nearly all

the cases of this condition the patients either had neglected to use the goggles provided or had not kept their heads behind the protective screen.

Minton discusses the extent of industrial injuries of the eye and their prevention.

W. ZENTMAYER.

VASCULAR CHANGES IN THE NORMAL FUNDUS PRODUCED BY SHORT WAVE THERAPY. E. TROVATI, Ann. di ottal. e clin. ocul. **64:** 603 (Sept.) 1936.

Patients were treated with two forms of apparatus, one delivering a wavelength of 30 meters and the other, a wavelength of 8 meters. Electrodes measuring 8 by 14 cm. were applied to the sides of the head for from twenty to thirty minutes. Observations were made before, during and after treatment. Ten patients with normal fundi were treated. All the patients showed evidence of vasodilatation, which was about the same with the two types of apparatus. This was seen either as hyperemia of the disk or as definite enlargement of both the veins and the arteries on the disk. It was seen after ten minutes of treatment and persisted until five hours afterward. No signs of hyperemia were visible after twenty-four hours. These signs of hyperemia suggest that the use of short-wave therapy would be dangerous in glaucoma but of possible value in certain ocular diseases due to abnormal vascular conditions. In such cases the results, which will be reported later, were encouraging.

S. R. GIFFORD.

Lacrimal Apparatus

PATHOLOGIC HISTOLOGY OF THE LACRIMAL GLAND. H. HERKEN, Arch. f. Augenh. **110:** 61, 1936.

The lacrimal glands were obtained at autopsy from one hundred and eight patients and examined histologically. Large amounts of fat were observed in the epithelial cells in the majority of cases; this does not represent a pathologic process. In senile glands pigment was frequently noted, together with the fat. In cases of tuberculosis, generalized infections, carcinoma and chronic diseases excessive amounts of fat were present.

In sixty patients there was definite lipomatosis, usually combined with atrophy of the cells of the gland. Forty of these patients were over 50 years of age. Atrophy of the gland can occur with or without replacement by connective tissue. All the patients over 50 years of age showed hyperplasia of connective tissue.

The lacrimal gland of the new-born does not contain any lymphatic tissue. Follicular collections of lymphatic tissue are found only later in life, and in atrophic glands this tissue is absent.

Acute inflammatory changes were observed in the glands of five patients with diphtheria. In one case of leukemia there was bilateral enlargement of the glands as in the Mikulicz syndrome.

F. H. ADLER.

Lens

ROENTGEN-RAY CATARACT. P. J. LEINFELDER and H. I. KERR, Am. J. Ophth. **19:** 739 (Sept.) 1936.

From an experimental, clinical and microscopic study, Leinfelder and Kerr arrived at the following conclusions:

"1. Nonprogressive cataract was the usual result of irradiation of the rabbit lens with ordinary therapeutic doses of roentgen rays.

"2. In equal dosage short roentgen rays were better tolerated than the long.

"3. Two children, after a latent period of two years, developed opacities in the lenses following roentgen-ray therapy.

"4. No opacities occurred in the lenses of adults when the eye was shielded.

"5. Small doses of roentgen rays did not affect the lens (case 2, right eye).

"6. Nonprogressive changes consisted of a posterior polar horizontal linear opacity with radiating rows of vacuoles.

"7. Microscopic examination of rabbit and human lenses showed subcapsular swelling and degeneration of lens fibers.

"8. Primary injury by roentgen rays appeared to affect the lens epithelium, the cells of which subsequently formed pathologic lens fibers.

"9. Early microscopic changes were seen at the equator, but with growth the damaged fibers extended to the posterior polar region.

"10. Anterior subcapsular and cortical changes indicated more severe damage that resulted in total opacity of the lens.

"11. In the later stage of the nonprogressive cataract the microscopic changes were greatest at the posterior pole.

"12. Clinical and histological studies indicated that progressive cataract did not invariably follow exposure of the human lens to roentgen rays."

W. S. REESE.

STAPHYLOCOCCUS TOXIN COMBINED WITH LENS EXTRACT AS A DESENSITIZING AGENT IN INDIVIDUALS WITH A CUTANEOUS SENSITIVITY TO LENS EXTRACT. E. L. BURKY and H. C. HENTON, Am. J. Ophth. **19:** 782 (Sept.) 1936.

Burky and Henton report two cases which show that the cutaneous reaction to lens extract can be changed from positive to negative by the intracutaneous injection of a mixture of staphylococcus toxin and lens extract. This change cannot be effected by the injection of lens extract alone. The evidence also points to a loss of ocular sensitivity, though this was not proved.

W. S. REESE.

DINITROPHENOL CATARACT. H. F. WHALMAN, Am. J. Ophth. **19:** 885 (Oct.) 1936.

This article does not lend itself to abstracting. It is a review of the use of dinitrophenol in which the methods of use, experimental and clinical, are described. Whalman then describes the type of cataract

resulting from the use of this drug. A tabulation of data for twenty-seven cases of cataract as to the age and sex of the patient, the dose, the loss of weight, the time of onset after treatment and other data is given. Whalman states that the operative treatment of this type of cataract is successful, and for patients up to the age of 45 he favors linear extraction.

W. S. REESE.

CATARACTA BRUNESCENS—STUDY OF THE NATURE OF THE COLORING SUBSTANCE. E. PUSCARIU and J. NITZULESCU, Brit. J. Ophth. **20:** 531 (Sept.) 1936.

The authors have met with this form of cataract 4 times in 1,357 cases of cataract. In only one of the four instances was the patient myopic. In all four cases the postoperative course was perfectly normal. The vision after extraction of the cataract remained low, making the prognosis for such cataracts less favorable than that for ordinary senile cataract.

The two lenses of one patient were chemically examined. A colored substance could be extracted with hot 3 per cent solution of potassium hydroxide. All chemical tests made tended to prove that this substance belongs to the so-called "Abnützungs pigment" (lipofuscin). The dopa reaction was negative. This finding does not exclude the possibility of an intermittent intervention of certain oxidative pigmentogenic enzymes, as advanced by Gifford and Puntenny.

Cataracta brunescens represents probably a far advanced stage of the process which produces usually yellow senile sclerosis of the lens. As in brown atrophy of the heart and of the muscles, it represents an accumulation of a pigment belonging to the group of lipofuscin.

W. ZENTMAYER.

POST-OPERATIVE DISTRESS IN CASES OF SENILE CATARACT. W. H. McMULLEN, Brit. J. Ophth. **20:** 657 (Dec.) 1936.

McMullen states that postoperative distress in cases of senile cataract is partly physical and partly mental and that the physical and mental factors react one on the other. The physical causes are pain in the back and abdominal discomfort, usually associated with flatulence. The causes of the pain and distress are discussed. A specially designed mattress with a cushion beneath the small of the back and a pillow beneath the knees may delay the onset and lessen the severity of the pain in the back. The author has found it impossible to prevent or relieve the pain in many cases so long as the patient is kept flat on the back. The abdominal pain and flatulence may be relieved by diet and drugs, but no measure is so effective as permitting the patient to be propped up in bed at an early stage and to be turned on his side and allowed to draw up his knees.

To relieve mental distress the patient should be brought to the operating room in as confident and optimistic a mood as possible. Any technic which unnecessarily prolongs the operation should be avoided.

Two chief causes of postoperative mental distress are immobility and the binocular bandage. Restraint is unnecessary if the eyes are well

shaded and a watchful nurse is employed. A binocular dressing is applied for at least two or three days unless the patient finds it very trying, mental derangement occurs or conjunctivitis develops, in which case only the eye that was operated on is bandaged, and a flap of gauze is placed over the other eye, which the patient can lift when desired. If no complications arise the patient is kept in bed only two or three days.

The article is well worth perusal by all ophthalmic surgeons.

W. ZENTMAYER.

Lids

OPERATIVE TREATMENT OF SPASMODIC ENTROPION. N. POCHISOW, Ann. d'ocul. **173:** 737 (Sept.) 1936.

An article on the operative treatment of spasmodic entropion by Poulard, published in the *Annles d'oculistique* in February 1935, was similar to an article by Pochisow published in January 1935 in the *Sovetskiy vestnik oftalmologii.* As Pochisow's method differs somewhat from the operation of Poulard he has thought it wise to refer again to this subject.

The operation consists of two parts: classic canthotomy and section of the external ligament lengthwise (the length of the tendon). It is important to obtain section of the ligament from the point of insertion at the external border of the orbit. Pochisow now does canthoplasty and uses two methods.

In the first method he places the sutures of the lower lid in such a way as to stretch it, by drawing upward, toward the palpebral conjunctiva. The second suture is placed so as to enter in a similar way and to cause an artificial entropion, which disappears in a few days, the lid becoming normal. In the second method the needle is introduced into the skin of the upper lid, the conjunctiva not being touched, and passed into the conjunctiva of the lower lid near the end of the cul-de-sac. It finally passes into the skin of the lower lid at a little higher level. By drawing the palpebral conjunctiva higher and outside and fixing it in this manner one prevents the possibility of recurrence.

There are three illustrative drawings.

S. H. MCKEE.

SURGICAL TREATMENT OF CONGENITAL COLOBOMA OF THE LIDS. J. CHARAMIS, Ann. d'ocul. **173:** 810 (Oct.) 1936.

Congenital coloboma of the lids, a rare malformation, was first described at Montpellier in a thesis of Mayer in 1808. A more thorough study was published for the first time in England by Wylde in 1862. Interesting are the reports by von Hippel in 1907 and Pagenstecher in 1912. Among recent reports the writer cites that of van der Hoeve in 1921.

A general description of coloboma and the different theories to account for it are given. Charamis describes a case in which he had followed the patient from birth. A girl was examined for the first time at the age of 6 months and again at the age of 9 years; no treatment had been carried out. The father had syphilis, and the child was given antisyphilitic treatment. There were no other congenital anomalies. The cornea was intact, although it was completely uncovered with the lids closed, so it was continually exposed and without protection.

The coloboma occupied nearly the whole width of the upper lid and extended practically to the limits of the tarsus. However, the movements of the upper lid were conserved despite this.

As regards the etiology, Charamis thinks that syphilis should be taken into serious consideration. This accords with the theory of van Duyse. The treatment of all palpebral colobomas should be surgical. Sometimes a simple drawing together of the border suffices; for those of greater width blepharoplastic operations are necessary. The correction in the case observed by Charamis is described in detail, and the article is illustrated with five photographs.

S. H. McKee.

Methods of Examination

An Illuminating Device to Be Used as an Attachment to the Binocular Corneal Microscope for Gonioscopy and Goniophotography. R. Castroviejo, Am. J. Ophth. **19:** 786 (Sept.) 1936.

This article describes an illuminating device that can be attached to a corneal microscope so as to permit gonioscopy, goniophotography and examination of the fundus by means of contact glasses. Photographs of the fundus were also obtained by this method.

W. S. Reese.

Infra-Red Photography of the Fundus of the Eye. E. Diaz-Caneja, Bull. Soc. d'opht. de Paris, January 1936, p. 55.

The history and evolution of the use of red-free light and of the infra-red rays in ophthalmology and photography are reviewed. Nineteen infra-red photographs are included in the article, and a description is given. A reading of the entire work is necessary to understand the article clearly.

L. L. Mayer.

Neurology

Emotional Factors in Mental Retardation: A Reading Problem. R. C. Hamill, Arch. Neurol. & Psychiat. **36:** 1049 (Nov.) 1936.

Hamill considers the emotional factor in reading difficulties, summing up his point of view in the following conclusion: "The fear of words can lead to evasions that interfere with learning to read. Looking ahead to avoid trouble may lead to difficulties with recognition and pronunciation of the word at hand. This may have some relationship to such mechanisms as reversal. Children, not knowing words, can be led into fears of meeting taboo words. Such fears lead to resentments and rejections of learning. Most taboo words have to do with natural processes of the body. Desire to know about these processes is inevitable. Expressions of that desire are frowned on. Words are the natural means of expression. Knowledge is feared and resented." Two cases of reading disability are cited in which Hamill believes the emotional element was sufficient to cause the difficulty. He also discusses the reading problem in relation to handedness and attempts to show a relationship between handedness and emotional conflicts.

R. Irvine.

Ocular Muscles

THE ACTION OF THE OCULAR MUSCLES. J. VAN DER HOEVE and C. O. ROELOFS, Arch. f. Augenh. **110:** 1, 1936.

Van der Hoeve and Roelofs used Volkmann's data on the origin and insertion of the ocular muscles with certain corrections, and with the aid of a coordinate system have calculated the position of the origin and insertion of each of the ocular muscles in relation to the point of rotation of the eye. This point lies 1.29 mm. back of the center of the eye. Although these values are relative, they give information regarding the action of each of the ocular muscles and their axes of rotation.

The results prove that the superior oblique muscle and the inferior oblique muscle are mutual antagonists and that for every ocular movement at least three ocular muscles come into play.

F. H. ADLER.

Operations

SOME OCULAR OPERATIONS. M. AMSLER, Schweiz. med. Wchnschr. **66:**1268 (Dec. 12) 1936.

The three procedures discussed are dacryocystorhinostomy, extraction of the cataract in its capsule and the operations for detachment of the retina. The West technic is reserved for the rhinologist. The operation of Toti as modified by Dupuy-Dutemps and Bourguet is for the ophthalmologist. Its indications are precise. It is limited to the correction of the obstruction of the nasolacrimal canal caused by chronic dacryocystitis. It aims to provide catheterization of the canal, removal of diseased tissues and restoration of function. It can be performed in forty-five minutes or less.

By extracting the lens in its capsule there is no necessity for awaiting the maturity of the cataract, and discission of the secondary membrane is eliminated.

Detachment of the retina was considered an incurable condition until the time of Gonin. Closure of the tear or hole is of the utmost importance to secure a successful result. The Paquelin cautery has been abandoned as an instrument of historical interest only. Galvanocautery, electrolysis and diathermy are now the technics of choice.

Ocular surgery has made rapid strides in recent times.

L. L. MAYER.

Orbit, Eyeball and Accessory Sinuses

A CASE OF "EXOPHTHALMIC OPHTHALMOPLEGIA WITH THYROTOXICOSIS." H. B. STALLARD, Brit. J. Ophth. **20:** 612 (Nov.) 1936.

A case of exophthalmos occurring in conjunction with unilateral partial ophthalmoplegia affecting the inferior rectus and inferior oblique muscles and associated with a basal metabolic rate of —4 per cent and mild general symptoms of thyrotoxicosis in a man 31 years of age is reported. After the administration on three successive days of 600 organon units (1,200 Schoeller units) of thyrotropic hormone there were a temporary rise in the basal metabolic rate, an increase in the

temperature and in the pulse and the respiration rate, increased exophthalmos, digital tremor and a perspiring, clammy skin. Later, despite tarsorraphy, it was necessary to enucleate the eye because of extensive destruction of the cornea..

The extra-ocular muscles were five or six times the normal size and were pale and fusiform. Histologically the muscle tissue showed interstitial fibrosis and chronic inflammatory infiltration by round cells. The muscle fibers exhibited no definite degenerative changes.

The clinical features of the case just reported—the relatively small degree of exophthalmos, the moderately increased pulse rate (from 64 to 90), the slight enlargement of the thyroid and the markedly defective action of the two extra-ocular muscles of the left eye on the same side as the greater degree of exophthalmos—conform to those described by Russell Brain under the diagnostic label of exophthalmic ophthalmoplegia thyrotoxicosis. These features, together with the low basal metabolic rate (—4 per cent) and the relatively slight general symptoms, suggest that possibly some factor other than the products of hyperactivity of the thyroid gland was responsible for the exophthalmos and the changes in the extra-ocular muscles. "The injection of thyrotropic hormone of the pituitary was in the nature of a clinical test and it produced such interesting events as a temporarily raised basal metabolic rate, increased temperature, pulse and respiration rate, increased exophthalmos, digital tremor and a perspiring, clammy skin. It may be that the degree of exophthalmos which developed after these injections of thyrotropic hormone would have been less in a case of a normal control."

W. ZENTMAYER.

PATHOLOGIC OCULAR HYPOTENSION. A. MAGITOT, Ann. d'ocul. **173:** 785 (Oct.) 1936.

In spite of imperfections the tonometer may be used for taking the normal tension, a high and a low limit being recognized. The normal tension is approximately 10 mm. of mercury, but one must not consider this an absolute standard. In other words, the ocular tension must always be compared with the general arterial pressure.

There are three influences which modify the ophthalmic tension: the local circulation, substances circulating in the blood and nervous influences. These influences may be found singly, but oftener they are combined.

In certain cases hypotension is due to congenital malformations; in others it is due to familial myopia, but hypotension in myopia is not the rule. Among the traumatic hypotensions are those that follow trauma in the ciliary region, burns of the limbus and contusions and those that are caused by surgical intervention on the globe. Hypotension in detachment of the retina is far from constant. It is frequent and does not appear to be linked to the myopia, and when it does appear it causes profound disorganization of the ocular membranes. In infections and toxemias, hypotension is frequent and here one meets the problem of hormonal influences.

Nervous influences constitute an important factor in hypotony, but what has been called transitory or essential ophthalmomalacia has become rare since the tonometer has come into use.

S. H. MCKEE.

RESTORATION OF THE EYE SOCKET WITH A THIERSCH GRAFT. LIN CHING-K'UEI, Chinese M. J. **50:** 1335 (Oct.) 1936.

The author describes in considerable detail six cases in which complete or partial plastic restoration of the eye socket was attempted. In five cases this was successful, and in one case failure resulted. The preoperative requirements, such as correction of any deformity of the lid, the elimination of pus-forming pockets and treatment of the margins of the lid, are stressed. As an anesthetic procaine hydrochloride, given by retrobulbar injection, was used.

The operation, the preparation of the mold and the taking of the graft are described in detail. No mention is made of the work of either Esser or Gillies.

The article is profusely illustrated, and the importance of plastic surgical procedures on the eye in China is emphasized.

S. H. McKEE.

The Pupil

PUPILLARY VARIABILITY IN ONE HUNDRED AND EIGHT SYPHILITIC PATIENTS. T. M. SHAPIRA and F. M. CRAGE, Am. J. Ophth. **19:** 891 (Oct.) 1936.

Shapira and Crage discuss the significance of the Argyll Robertson pupil and anisocoria. They give the following summary:

"In this series of 108 syphilitic patients, 32 patients had pupillary irregularity in both eyes; in 8 patients the pupils were irregular in one eye.

"Pupils were unequal in 42 patients.

"Pupils did not react to accommodation in 19 patients.

"Reaction to light was absent in 33 patients.

"Definite typical Argyll Robertson pupils were present in 17 patients.

"Five patients showed reflex pupillary rigidity.

"Considering a miotic pupil as being less than 2.5 mm., miosis was present in both eyes in 15 patients, and in one eye in 16 patients."

W. S. REESE.

Physiology

DISTRIBUTION OF INTENSITY OF THE LIGHT SENSE IN THE PERIPHERAL PARTS OF THE RETINA. L. N. GASSOWSKY and N. J. NIKOLSKAYA, Sovet. vestnik oftal. **9:** 123, 1936.

Gassowsky and Nikolskaya measured in sixteen subjects the absolute threshold for light of the periphery of the retina and the corresponding sensitivity of the retina at separate points (inverse to the threshold) on both sides of the fovea, in the horizontal meridian. The threshold for light was determined for point stimuli, with a visible angle of 2 minutes and 28 seconds at 4, 6, and 8 degrees and up to 20 degrees from the fovea. The basic parts of the experiment were white and red illuminated dots which could be seen by the observer through the slit of a screen; the intensity of their illumination could be regulated by

the observer. The observer remained in complete darkness for one hour and a half before he could start registering his findings with the illuminated dots. Further details of the experiment are given in detail.

The authors illustrate with a table and diagram that the absolute threshold for light is decreased up to 10 degrees from the fovea on both the nasal and the temporal side. The sensitivity of the retina at 10 degrees from the fovea is twice as great as at 4 and 20 degrees.

O. SITCHEVSKA.

Retina and Optic Nerve

INHERITANCE OF OPAQUE NERVE FIBRES IN THE RETINA (PAPILLA LEPORINA). E. A. COCKAYNE, Brit. J. Ophth. **20:** 569 (Oct.) 1936.

After describing the development of opaque nerve fibers and enumerating the various congenital anomalies which have been found associated with this condition, Cockayne gives the pedigree of a family in in which ten members are affected: In four of them the condition is bilateral; in six, unilateral. In all of them the area covered by the opaque fibers is small. Five males and five females are affected, and in two of each sex the condition is bilateral. Of those examined in the sibships containing affected members, ten (five males and five females) are affected, and ten (four males and six females) are normal. Thus the ratio of affected to normal persons is 1:1, and descent is direct in every case. Males and females show the condition to an equal extent, and it is bilateral in the same number of females as of males. Both these observations are contrary to expectation if it is sex-linked. It is true that there is no instance of transmission of the abnormality by a male, but only one of the affected males has children, and only one of these children is old enough to show the defect.

Though the pedigree does not disprove Kisö's hypothesis that the condition is sex-linked, its inheritance is quite in accord with that of autosomal dominants, and since autosomal dominants are common and sex-linked dominants are of extreme rarity in man it is probable that opaque nerve fibers fall into the former rather than into the latter group.

W. ZENTMAYER.

BIPOLAR ELECTROLYSIS IN THE TREATMENT OF DETACHMENT OF THE RETINA. HUDELO, Bull. Soc. d'opht. de Paris, October 1935, p. 642.

Electrolysis for the treatment of detached retina is not new, especially the unipolar method, which was used before 1895 by Abadie, Gillet de Grandmont, Moll, Terson and, more recently, by Vogt. The bipolar method was employed in 1893 by Schoeler, but his technic was entirely different from the original simple and efficacious method of Hudelo.

The objection to the unipolar method is that it coagulates only at the level of the electrode in a single point, and the electrolysis is limited to feeble intensities in order not to damage the vitreous. The bipolar method fixes the globe and coagulates an area in the form of a crown. Platinum-iridium electrodes separated and insulated from each other at a distance of 3 mm. may be used without complete removal of the

conjunctiva over the area as done by Arruga. The entire technic is described. Adequate ophthalmoscopic control is always present. Five observations are reported.

L. L. MAYER.

FIVE OBSERVATIONS OF TRAUMATIC LESIONS OF THE MACULA. C. COTELA, H. LAGRANGE and L. BONHOMME, Bull. Soc. d'opht. de Paris, December 1935, p. 804.

The authors report in detail five cases in civilians in which trauma caused lesions of the macular area, and compare these traumas with traumas occurring during the war period. The observation that any type of trauma to the region of the head may cause macular lesions, as this area is seemingly less resistant than others, is in accord with the findings of F. Lagrange during the war period. Delay in finding macular lesions is common and therefore has an important medicolegal interest.

L. L. MAYER.

LOCAL SURGICAL TREATMENT OF PAPILLARY STASIS. GOMEZ-MARQUEZ, Bull. Soc. d'opht. de Paris, December 1935, p. 814.

A surgical method to prevent the occurrence of atrophy of the optic nerve due to edematous pressure in papilledema is presented. Canthotomy is done on the temporal side, and then a vertically curved incision enables one to make an exposure whereby, when the external rectus muscle is divided, the optic nerve is readily made accessible. Tenon's sheath and connections with the dura are slit by the assistance of an aneurysm needle. Gomez-Marquez has practiced the operation on cadavers but has not used it as yet on living human subjects. Excellent drawings depicting the procedure accompany the article. It is believed that the chances of infection of the brain are much less than with a cranial operation and that such an operation may be especially useful in cases of increased intracranial pressure in which localization is not exact or in which a more formidable operation is not warranted.

L. L. MAYER.

NEW PATHOGENIC AND THERAPEUTIC TRENDS IN TABETIC ATROPHY OF THE OPTIC NERVE. H. ARRUGA, Arch. de oftal. hispano-am. 36: 365 (July) 1936.

This paper is based on Arruga's clinical experience in connection with Lauber's theories on tabetic atrophy of the optic nerve. These concern the general arterial hypotony and the relatively increased intra-ocular tension in this condition. The indicated treatment is raising the general tension and lowering the intra-ocular tension. Although the number of cases in which Arruga applied the treatment was fifteen, he reports only six, the others having been observed too short a time or incompletely. Among these a fairly satisfactory result was observed in three. Apart from the antisyphilitic treatment, general treatment consisted of dietetic measures and the use of strychnine, caffeine, epinephrine and ephedrine; the local treatment consisted in the administration of pilocarpine and, occasionally, cyclodialysis.

C. E. FINLAY.

DIAGNOSTIC AND PROGNOSTIC VALUE OF EXAMINING THE BLOOD VESSELS OF THE EYE IN HYPERTENSION. N. A. PLETNEVA, Sovet. vestnik oftal. **9**: 3, 1936.

Hypertension, according to Pletneva, is on a neurohumoral basis; it is a form of the functional vegetative neurosis; a spasm of the precapillary system is produced, which leads later to sclerotic changes in the precapillary vessels. Pletneva examined 104 patients with hypertension in the Institute of Functional Diagnosis. The ages of the patients varied from 20 to 80 years. The systolic blood pressure in all the patients was above 170 mm. of mercury. Not only the retinal but also the conjunctival and limbal blood vessels were examined. Eighteen patients suffered from albuminuric retinitis with spasm of the blood vessels. Seventeen of these patients had nephrosclerosis, and ten of them died. Eight patients had spasm of the retinal vessels; the arteries were narrow, some appearing like silver wires, with a marked difference in caliber between the arteries and the veins. Ten patients suffered from unilateral thrombosis of the central vein without the kidneys being affected. Salus' symptom (indentation of veins by arteries at the crossing) was observed in 50 per cent of the patients. Thirty patients showed this symptom while the kidneys were unaffected. The so-called angiospastic retinitis of Volhard is met with in "pale" hypertension (i. e., there is a spasm of the peripheral vessels), which indicates a pathologic condition of the precapillary system of the peripheral vessels, the prognosis of which is poor.

With the slit lamp Pletneva examined the capillaries of the corneal limbus in 91 patients. Twenty-eight patients suffering from "pale" hypertension presented complete obliteration or absence of the fine limbal net. In some capillaries which retained their patency an engorgement of the venous knee could be seen. The blood circulation was irregular. Nineteen of these patients showed capillary changes coinciding with those of the retinal vessels. All these patients suffered from nephrosclerosis. In "red" hypertension (i. e., the peripheral vessels were dilated) the limbal capillary net was engorged and hyperemic; several aneurysms and hemorrhages were noted.

Pletneva draws the following conclusions: The prognosis of hypertension can be made by careful examination of the retinal vessels and of the capillaries of the corneal limbus. Angiospastic retinitis is usually met with in patients who have sclerosis of the blood vessels of the kidneys, brain and heart; it is prognostic for malignant hypertension. An ophthalmologic examination should always be made in hypertension, as the changes in the blood vessels of the eye indicate the condition of the peripheral blood vessels of other organs: heart, brain and kidneys.

O. SITCHEVSKA.

Trachoma

TREATMENT OF TRACHOMA IN CHILDREN. M. ZACHERT, Rev. internat. du trachome **13**: 177 (Oct.) 1936.

Trachoma in children is generally benign and is rarely associated with severe complications of the cornea or lid. Though children have a lesser immunity to the contagion than adults, their resistance to its

progress is greater. The granulations should be crushed individually and in several sittings. Frottage with chaulmoogra oil is well tolerated.

J. E. LEBENSOHN.

TRACHOMA THERAPY WITH QUININE BISULPHATE. E. SELINGER, Chinese M. J. **50:** 1449 (Oct.) 1936.

When applied to mucous membrane, quinine bisulfate acts as an astringent, is mildly anesthetic and, being an alkaloid, penetrates through the mucous membrane. It is a bactericide, and because it is a protoplasmic poison it destroys leukocytes and lymphocytes and inhibits the invasion of the tissues by these elements. The bisulfate is used because it is more water-soluble than most of the other salts.

The results obtained indicate that this form of treatment is shorter than that with copper sulfate and chaulmoogra oil, that corneal complications are favorably influenced by it and that the final visual results are better than those obtained with other forms of therapy for trachoma.

S. H. MCKEE.

Tumors

PAPILLOMATA AND OTHER TUMORS OF THE LIDS AND THEIR TREATMENT WITH THE ELECTROLYSIS NEEDLE. J. MINTON, Brit. J. Ophth. **20:** 624 (Nov.) 1936.

This is a plea directed to the ophthalmic surgeon for the use of the electrolysis needle for the removal of certain tumors of the lids.

The galvanic current is required. The machine now used in the epilation of lashes may be employed. A curved or straight steel needle is preferred. A current of from 3 to 4 milliamperes is necessary for destruction of the base. In the removal of a papilloma the needle is made to transfix the growth in several directions. The needle is kept in position at each transfixion only a few seconds. The growth is then snipped off above the cauterized base. If the needle is left in too long or if the current is too strong the surrounding healthy tissue will be destroyed and a scar may result. Other growths require certain modifications of the technic just described.

W. ZENTMAYER.

TUMOR OF THE OPTIC NERVE (NEUROPERINERVOSA GLIOMATOSA). F. ROSSEAU, A. DOLLFUS and G. ARDOIN, Bull. Soc. d'opht. de Paris, October 1935, p. 629.

For the past two years it had been noted that a boy of 7 years had prominence of his right eye. From time to time there had been a spontaneous receding to normal. The exophthalmos was not reducible; excursion was nil upward and was limited in all other directions, mostly laterally. The lids could be closed, and the cornea was well protected. The papilla showed stasis, with atrophy of the superior portion of the optic nerve. Pupillary reaction was absent, although the patient counted fingers at 30 cm. The sinuses were not involved. The tumor is pictured grossly, and several photomicrographs show the nature of the tissue cells. The tumor entirely surrounded the optic nerve.

L. L. MAYER.

CONGENITAL PIGMENTED CYSTS OF THE IRIS AND THE CILIARY ZONE; VESTIGE OF THE VASCULAR MEMBRANE OF THE LENS; PERSISTENCE OF THE HYALOID ARTERY. F. BADEAUX, Bull. Soc. d'opht. de Paris, January 1936, p. 68.

The clinical rarity of congenital pigmented cysts of the iris and the ciliary zone and the infrequency of reports of the condition, as studied by means of the slit lamp or by pathologic examination, prompted this article. In a patient 10 years of age, on dilatation of the left pupil a mass was found back of the iris between 6 and 9 o'clock. Pictures taken with the use of the slit lamp are reproduced. The history of such cysts is reviewed, and drawings are shown to demonstrate the embryology of this type of cyst. Badeaux believes that such cysts are caused by doubling of the epithelium of the uveal layer and subsequent incomplete absorption. Surgical intervention is indicated if visual acuity is lowered. If such a growth is watched over a long period its benign nature is confirmed.

L. L. MAYER.

Uvea

UVEITIS ASSOCIATED WITH ALOPECIA, POLIOSIS, DYSACOUSIA AND VITILIGO. T. H. LUO, Chinese M. J. **50:** 1409 (Oct.) 1936.

The type of uveitis described is bilateral, develops rapidly and reaches its height in a short time. Often it is preceded by such prodromal symptoms as headache, drowsiness, nausea and a feeling of heaviness about the head. In most of the Japanese reports, lesions of the choroid are reported. Late in the course of the disease the ocular picture is chiefly that of inflammation of the anterior part of the uvea, with the formation of seclusio and occlusio pupillae, which prevent ophthalmoscopy and may conceal changes in the fundus.

The alopecia and poliosis which occurred in practically all the reported cases made their first appearance about from three weeks to nine months after the onset of the disease. The vitiligo, dysacousia and tinnitus were present in a little over half the cases. Tinnitus with temporary impairment of hearing was noted early in the course of the disease, while vitiligo appeared shortly after the development of the alopecia and poliosis.

The etiology of the disease is unknown; consequently the treatment is nonspecific. The grave prognosis should always be borne in mind.

S. H. MCKEE.

Vitreous

CLINICAL STUDIES ON THE VITREOUS: THE DETACHMENT OF THE CHOROID OF LEAKING EYES. K. LINDNER, Arch. f. Ophth. **135:** 462 (July) 1936.

If the aqueous is aspirated from the eye of a cadaver, a fluid which is obviously derived from the vitreous slowly refills the chamber. The vitreous gives off this fluid under the influence of pressure created by the forward displaced lens. The same mechanism, according to Lindner,

applies to the living eye. In a leaking eye (with a corneal or vitreous fistula) no such pressure is available. In leaking eyes with choroidal detachment the volume of the vitreous is greatly reduced; it must have given off water, not under the influence of any pressure, but under the influence of chemical forces. The regenerated, abnormal aqueous which seeps through the vitreous in these fistulating eyes contains the agent that makes the vitreous shrink. This shrinking is the real cause of choroidal detachment. In the eyes of young persons the vitreous detaches itself from the retina more easily than in the eyes of older persons. That is the reason why choroidal detachment occurs only in older persons. (See also Lindner: *Arch. f. Ophth.* **135:** 332, 1936.)

P. C. KRONFELD.

ANATOMIC STUDIES OF POSTERIOR SEPARATION OF THE VITREOUS. L. SALLMANN, Arch. f. Ophth. **135:** 593 (July) 1936.

Lindner's views concerning the mechanism of retinal detachment and the clinical observations of posterior separation of the vitreous made with the slit lamp by those belonging to Lindner's school have brought the anatomy of this condition to attention again after von Hippel in 1908 had practically settled the until then very active discussion concerning it. Sallmann has examined anatomically twelve fresh human eyes with various clinical conditions, studying them shortly after enucleation and without the use of a fixative. In two of these eyes posterior separation of the vitreous was macroscopically visible; in two others macroscopic combined with histologic examination made the diagnosis of the condition certain. The usual fixing and hardening process often produces artificial posterior separation, in which case not a trace of albuminous fluid can be found between the retina and the separated vitreous. A true separation is exaggerated by the fixing process. To differentiate between the hyaloid membrane and a border layer produced within the vitreous by partial liquefaction, the pyridine method of Redslob is very useful, because it produces a well defined opacity of the hyaloid membrane only.

P. C. KRONFELD.

Sympathetic Ophthalmia

A CASE OF SYMPATHETIC OPHTHALMIA FOLLOWING GONORRHEAL CORNEAL ULCER, OBSERVED FOR NINETEEN YEARS. GROENOUW, Klin. Monatsbl. f. Augenh. **96:** 742 (June) 1936.

Groenouw refers to Peters' report in the literature for 1919-1935 on sympathetic ophthalmia and describes the case of a man aged 43 who suffered a rupture of a gonorrheal corneal ulcer and prolapse of the iris in 1911. The second (right) eye became inflamed two months later; the irritation disappeared after local treatment and enucleation of the left eye. However, recurrences were observed after one, seven, eight and nine years and in a mild form thereafter. The eye was found to be free from any symptoms, and vision at the last examination, in 1930, was practically normal. Sudden death followed soon after this examination. The blood count, which is supposed to show changes indicative of sympathetic ophthalmia, showed a decided decrease of polymorpho-

nuclear leukocytes and an increase of eosinophils and mononuclear lymphocytes. Small, round, white dots, characteristic of sympathetic choroiditis, were observed in the peripheral portion of the choroid. Groenouw's case is the third case of this type in which these dots disappeared. The author stresses the fact that there were numerous recurrences, although recovery was promptly obtained in the beginning, and that nevertheless the condition of the eyes allowed strenuous visual activity.

K. L. STOLL.

Therapeutics

GLYCEROL TRINITRATE (NITROGLYCERINE) IN THE TREATMENT OF HEMERALOPIA (NIGHT-BLINDNESS). R. DE R. BARONDES, Brit. J. Ophth. **20:** 528 (Sept.) 1936.

To the usual condition to which hemeralopia is ascribed, Barondes adds a circulatory disturbance of dysfunction in which the retinal arteries and capillaries remain in a spastic state. This spasticity is observed to be more pronounced in the vessels in the fringe of the retina, that part of the retina under faint illumination. The nerve endings in the light-perceiving area—the retinal rods—are dependent on a proper supply of food for the regeneration of the visual purple, the "night-seeing agent."

Barondes gives the ophthalmic finding in five adults with hemeralopia as a constricted, blanched appearance of the peripheral portions of the retinal arteries, with more or less venous hyperemia and congestion.

Nitroglycerin proved to be the most effective dilator of these spastic vessels, although the nitrites gave almost comparable results. It is suggested that vasodilator drugs might be administered to those working more or less continuously in the dark, to whom it is especially important to be able to see in darkness, as spies in wartime, navigators and those aviators who do night flying.

W. ZENTMAYER.

HYPODERMIC INJECTIONS OF CAROTENE IN THE TREATMENT OF PHLYCTENULAR CONDITIONS AND VITAMINE A DEFICIENCY DISEASES IN OPHTHALMOLOGY. M. C. TAI, Chinese M. J. **50:** 1453 (Oct.) 1936.

Cod liver oil possesses an unpleasant smell and is difficult to preserve from oxidation. Eggs, milk and butter are sometimes not well digested by certain patients. Vegetables are seasonable products and, even when available, may cause gastro-intestinal upsets. Carotene in ampules, administered hypodermically, obviates all the difficulties of oral ingestion. The only contraindication to its use is cirrhosis of the liver. Rigid asepsis should be observed in administering it, otherwise a moderate degree of dermatitis may develop. Carotene is inexpensive and is available in large amounts. It is produced in China and is therefore easily obtained in that country and may be widely used to great advantage in treating certain ocular conditions there.

S. H. MCKEE.

News and Notes

SOCIETY NEWS

Annual Congress of the Ophthalmological Society of the United Kingdom.—The following program was presented at the annual congress of the Ophthalmological Society of the United Kingdom, held from April 29 to May 1, 1937:

"Prognosis in Papilledema," Dr. Gordon M. Holmes, president.

"Retinal Circulation, Changes in Metabolic Disease," Dr. A. E. MacDonald, Toronto, Canada.

"Some Observations on Katholysis in Treatment of Retinal Detachment," Mr. R. Foster Moore.

"Criticisms and Comments on Newer Methods of Treating Detachment of the Retina," Mr. J. Cole Marshall.

"The Internal Limiting Membrane of the Retina," Mr. E. Wolff.

"Importance of the Near Balance Test," Miss Margaret Dobson.

"Pathogenesis of the Melanomata," Mr. E. F. King.

"Principles and Practice of Ocular Screening," Mr. G. F. Alexander.

"Clinical and Microscopic Appearances of Experimental Grafts of Scleral Tissue onto a Rabbit's Cornea," Mr. J. W. Tudor Thomas.

"Atropin Irritation," Mr. Fred Ridley and Dr. Ian Maclean.

"Innervation of Ocular Muscles and the Mesencephalic Root of the Fifth Nerve," Prof. H. Woollard.

"Spontaneous Cure of Retinal Glioma," Mr. Montague Hine.

"Visual Purple," Dr. R. J. Lythgoe and Dr. C. F. Goodeve.

"Exophthalmic Ophthalmoplegia," Dr. W. Russell Brain.

"Exophthalmic Ophthalmoplegia: Effect of Prostigmin in Two Cases," Prof. F. R. Fraser.

"Case of Exophthalmic Ophthalmoplegia," Mr. H. B. Stallard.

"Involvement of the Optic Nerve in the Demyelinating Diseases," Dr. J. Purdon Martin.

There was also an open discussion on the rarer forms of keratitis.

The president of the society and the medical staff of the National Hospital for Diseases of the Nervous System arranged a demonstration of cases of ocular palsies, disturbances of the visual field, ophthalmoscopic conditions and pathologic specimens of neurologic and ophthalmologic interest.

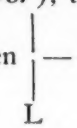
The following officers were elected for the year 1937-1938: president, Gordon M. Holmes; vice-presidents, T. Harrison Butler, R. Affleck Greeves and Percival Hay; treasurer, Sir Arnold Lawson; council, O. Gayer Morgan, Sir Stewart Duke-Elder, Ida Mann, A. Macrae, R. C. Davenport and J. D. M. Cardell; secretaries, H. B. Stallard and L. E. Savin.

GENERAL NEWS

Summer Courses in Ophthalmology and Otolaryngology in Vienna, Austria.—The twelfth intensive European summer courses in ophthalmology and otolaryngology, under the direction of Dr. George

W. Mackenzie, will be given in Vienna, Austria, during the summer of 1937. The course in ophthalmology includes microscopic anatomy and pathology of the eye; external diseases of the eye, with an exhibition of interesting cases; ophthalmoscopy, with an exhibition of cases; ward work demonstrating the treatment of patients, including the postoperative treatment; operations on pigs' eyes, and courses in the ocular muscles and slit lamp examination. The group leaves New York on July 10 and returns on September 4. Further information may be secured from Dr. Mackenzie, 269 South Nineteenth Street, Philadelphia.

CORRECTIONS

In the article by Prof. A. Bielschowsky entitled "Application of the After-Image Test in the Investigation of Squint," which appeared in the March issue (**17:** 408, 1937), the diagram in the last line but one on page 418 should have been  R. And the word "crossed," at the end of the fourth line on page 419, should read "uncrossed."

In the article by Dr. Samuel P. Oast, "Scleromalacia Perforans: Report of a Case," in the April issue (**17:** 698, 1937), two errors occurred. In the sixth line on page 700, "lymph nodes" should be inserted after "preauricular," and in the twentieth line on the same page, "eye" should be omitted after "neither."

Society Transactions

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Jan. 18, 1937

JOHN H. DUNNINGTON, M.D., *Chairman*

LEGRAND H. HARDY, M.D., *Secretary*

DACRYOCYSTORHINOSTOMY—TECHNIC AND RESULTS. DR. LOREN P. GUY.

A brief history of dacryocystorhinostomy from 1724 to the present was given. The indications, contraindications and complications were enumerated. A simple technic, requiring less than thirty minutes to perform, was described. Of the first 15 cases in which this operation was performed, success was reported in 13.

DISCUSSION

DR. WEBB W. WEEKS: Of all the procedures used to accomplish drainage of tears, the one that accurately approximates the edges of the wound and leaves the tissues in normal relationship seems the correct surgical operation. Dupuy-Dutemps and Bouget not only insisted on the contraindications mentioned in Dr. Guy's report, particularly the patency of the puncta and canaliculi, but also emphasized that the operation should be done when inflammation of the tissues was absent.

Since my report in 1932 of 8 cases in which the procedure previously described was carried out, it has been done in 6 additional cases. During the past year Dr. Wendell Hughes has carried out this procedure on 7 patients in our service at the New York Eye and Ear Infirmary. In 1 case the edges of the wound could not be sutured because of friable tissue. In another case the lower one third of the sac was carried through the mucous membrane of the nose and left in situ. All these patients have done well except 1, in whom the lower canaliculus was partially slit some years previously, with resultant impairment of function of this lacrimal drainage system.

According to the French procedure anesthesia is produced by the subcutaneous injection of a 2 per cent solution of procaine hydrochloride plus a 1 per cent solution of neosynephrin hydrochloride along the course of an incision in the skin extending to the dome of the lacrimal sac above and to the neck of the lacrimal sac below. In addition, the branch of the nasociliary nerve passing through the anterior ethmoid foramen is blocked, and the mucous membrane in front of the middle turbinate is raised by the same solution. The latter step protects the nasal mucous membrane during the removal of bone.

ANISEIKONIA WITH NO REFRACTIVE ERROR. DR. WENDELL L. HUGHES.

The relative size of the extra-ocular images in a group of 43 patients with symptoms that were thought to be ocular in origin was determined. In 14 the images were equal. In 29 they were asymmetrical.

The result in 14 cases in which complete data were obtainable was reported. Relief was obtained in 10 by the wearing of iseikonic zero power lenses designed to correct the aniseikonia present. It has been shown that the presence of aniseikonia is independent of any refractive error.

DISCUSSION

DR. JOSEPH I. PASCAL: I noticed that about 91 per cent of one group of patients with aniseikonia and photophobia were relieved by iseikonic lenses. It is known that ordinary white glass absorbs about 10 per cent of the light. Is there any possibility that the photophobia was largely relieved by the reduction of this 10 per cent of the light?

DR. WENDELL L. HUGHES: The question of photophobia is an interesting one, and I do not know exactly how to explain it, but the 91 refers to the percentage of patients complaining of photophobia who had aniseikonia. Most of the patients had been wearing lenses of minor strengths previously, and the photophobia had been unrelieved. The photophobia was relieved when the images were made symmetrical in size and were able to be fused easily.

CATARACT DUE TO DINITROPHENOL. DR. CLYDE MCDANNALD.

A case of cataract due to dinitrophenol was reported. The patient complained postoperatively of blurred vision, which was overcome by squinting. Transillumination demonstrated scattered areas of atrophy of the pigmented layer of the uvea. There were similar small rarefied areas in the eye on which no operation was performed. It was suggested that the rarefaction of the uveal pigment may be a factor in a toxemia causing opacities of the lens after the ingestion of dinitrophenol.

USE OF CELLULOSE ADHESIVE TAPE FOR RETAINING DRESSINGS FOR THE EYE. DR. DONALD BOGART.

A cellulose adhesive tape has been used at the New York Eye and Ear Infirmary for eighteen months for approximately 2,500 patients. It has the following advantages:

1. It retains dressings for the eye in place well and is easily removed.
2. It is less disfiguring to the patient than ordinary adhesive tape.
3. It is available in a large dispenser, which allows the cutting of pieces with one hand, thus promoting speed.
4. It is very much less expensive than ordinary tape used for dressings for the eye.
5. It usually leaves no residue on the face.
6. It is waterproof.

Scotch tape has the following disadvantages:

1. In warm weather the small roll may be difficult to "start."

2. In 5 of the 2,500 patients a mild dermatitis due to the tape developed. Patch tests on 3 of these demonstrated sensitivity to the tape or its gum.

SPONTANEOUS RUPTURE OF VENOUS ANGIOMA OF THE ORBIT. DR. WILLIAM B. DOHERTY.

In 1910, when the patient was 17 years of age, she stated that while she was working suddenly something "burst" in her right eye. There was intense swelling; the lids were very ecchymotic; the conjunctiva ballooned up with large subconjunctival hemorrhages, and the eyeball protruded. The patient was taken to the New York Eye and Ear Infirmary, and a Krönlein operation was performed by Dr. Emil Gruening. The orbit was found to be full of clots of blood, and a diagnosis of "idiopathic hematoma of the orbit'" was made.

During March 1923, about thirteen years later, a similar condition occurred, and the patient was admitted to the Bellevue Hospital in the service of Dr. Charles H. May. At that time the picture was almost identical to that seen in 1910, the patient showing marked ecchymosis of the lids, intense chemosis of the conjunctiva, and exophthalmos of at least 4 mm. Haziness of the cornea reduced the vision to counting fingers at 10 feet (3.3 meters). After recovery there were limitation of motion of the eyeball in all directions, partial symblepharon in the lower lid and marked evidences of a venous angioma. The angioma manifested itself in the lower lid, simulating a bag of blue earthworms which seemed to shine through, and the entire mass could be palpated by the fingers. The patient had a series of treatments with the roentgen rays and radium, given by Dr. G. Allen Robinson, and there has been a gradual diminution of the evidences of the tumor. Vision of the right eye was 20/50 with a —1.00 D. sph. ⊂ —1.00 D. cyl., ax. 90, and that of the left eye was 20/20 with a —1.00 D. sph. ⊂ —1.00 D. cyl., ax. 75. The fundus showed an area of choroidal atrophy; a roentgenogram of the orbit did not reveal any bony erosion, and the Wassermann reaction was negative. The fields presented a concentric contraction of about 15 degrees. The left eye, with the exception of the error of refraction, was normal.

SCLEROMALACIA PERFORANS. DR. SAMUEL OAST.

This paper was published in full in the April 1937 issue of the ARCHIVES, page 698.

DISCUSSION

DR. ALGERNON B. REESE: While inflammation of the sclera usually produces fibrosis which may lead to great thickening of the coat, rarely, as apparently Dr. Oast's case illustrates, can it lead to a nonreparative process and thereby leave a rarefied area. One ordinarily thinks of scleritis as affecting primarily the anterior part of the globe, because only this portion can be viewed. Inability to recognize inflammation in the posterior portion of the sclera was brought home to me forcibly some time ago. A patient had severe pain in one eye, for which no cause could be found. Finally a rise in the intra-ocular pressure was detected, and the feeling was that perhaps unrecognized glaucoma had been the cause of this pain, so a trephine operation was done. The intra-ocular pressure was controlled, but the pain continued to be as severe

as before. Finally the eye was enucleated because of the pain. Microscopic examination showed inflammation of the posterior portion of the sclera, with thickening of the sclera from three to four times that normally seen.

Dr. Lewis Webb Crigler: About fifteen years ago I had occasion to observe an elderly woman suffering from arthritis deformans. During the course of the disease low grade scleritis developed that involved the entire circumference of the eyeball adjacent to the limbus. The tissues seemed to undergo calcareous degeneration resulting in complete destruction of the entire thickness of the sclera, save a few fine trabeculae which seemed to hold the cornea and sclera together. The tension never rose sufficiently to cause protrusion of the unsupported choroid and ciliary body, which were plainly visible.

The patient died before it was deemed necessary or expedient to remove the eye; thus a rare and valuable specimen was lost. This case appears to come under the classification described by van der Hoeve, in which articular rheumatism was a complicating factor. The characteristic difference in the pathologic features in the two cases just described appears to be that in the one a degenerative process was present, and in the other, a postinflammatory one.

Glaucoma and Sympathetic Ophthalmia. Dr. Bernard Samuels.

This paper will be published in full in a later issue of the Archives.

Pathologic Conditions of the Orbit—Roentgen Studies. Dr. Irving Schwartz.

Roentgenography of the orbit consists, for the most part, in a careful study of shadows of the bony structures. Slight deviations in position or density may be of decisive diagnostic significance. In some cases roentgen studies may be the only means of deciding whether a disease process is within the orbit or in the periorbital structures, whether it is inflammatory or neoplastic, and, if it is the latter, whether the neoplasm is malignant or benign.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Jan. 21, 1937

Charles R. Heed, M.D., *Chairman*

Alexander G. Fewell, M.D., *Clerk*

Exhibition of a Case of Spontaneous Reattachment of the Retina. Dr. William Zentmayer.

This patient is being presented in connection with Dr. Buchanan's case because of complete spontaneous replacement of the retina following a detachment that was probably exudative in origin.

Vision of the right eye became distorted in April 1930. The first examination was made in September of that year, when it was found that vision equaled 6/15 and the upper part of the field of vision was lost. There was separation of the retina, which began at about the horizontal meridian of the fundus, and extended forward to the ora serrata. There was already marked retinitis striata, a white band demarcating the limit of the separation. The anterior part of the retina was thrown into small folds, and on the temporal side, well forward, there was an area of choroiditis about which there were a half dozen or more gray, almost globular bodies, each about three times the diameter of the central artery of the retina, similar in appearance to the lesions seen in sympathetic ophthalmitis. There were a few striae in the posterior cortex.

By the latter part of October the bodies had disappeared. It was not until August 1932 that it was noted that the retina was entirely back in place and the field was again full. The picture is now one of sequelae of the retinochoroiditis, with the unusual development of retinitis striata. Vision is 6/9.

A Case of Detachment of the Retina with Spontaneous Reattachment. Dr. Mary Buchanan.

Miss C. B. R. came to the ophthalmic clinic of the Woman's Hospital in March 1920, five weeks after her discharge from the Mount Sinai Hospital when convalescent from an attack of influenza. She was practically blind, since her right eye showed a detached retina, and her left, a cataract. After six weeks' treatment with a vaccine prepared from mixed influenza bacilli her vision improved to 5/30 in the right eye and 5/60 in the left. On August 9 the cataract was extracted from the left eye, and after this vision with a —2.00 sph. ⊂ —2.50 cyl., ax. 85 registered 5/15. In 1925 a cataract was extracted from the right eye, but in 1926, after the extraction of several teeth without the use of gas, the right eye showed an intra-ocular hemorrhage, and it had to be enucleated. In the meantime vision of the left eye became rapidly reduced. In the spring of 1936 the patient reported that there was something hanging over the central sight, and vision was gone. The ophthalmoscope showed a white wall, but no details of the fundus could be seen. Shortly after trauma to her left eye in August she found that she could see better toward the temporal side. Her field, which was taken on October 21, showed a 60 degree boundary below the horizontal line; the field extended 15 degrees above and 20 degrees to the nasal side. Vision was 1.5/60. On Jan. 18, 1937, a large staphyloma and two large patches of sclera could be seen at the nasal side.

A Case of Uveoparotitis in a Thirty-Two Year Old Negro. Dr. Alexander G. Fewell and Dr. Luther Kauffman.

A 32 year old Negro had uveoparotitis of about eight weeks' duration. The following features were present: markedly enlarged and indurated parotid glands on each side, facial paralysis on the right and some of the features of uveitis in each eye. Roentgen examination of the chest revealed thickening of the hilus shadows. The blood count revealed leukopenia and eosinophilia.

ILLUMINATION. DR. ALFRED COWAN.

Ophthalmologists should be familiar with the problem of artificial lighting and in agreement with illumination engineers in their endeavor to bring about higher intensities and proper arrangements of lighting fixtures.

DISCUSSION

DR. WILLIAM ZENTMAYER: Since Dr. Cowan has told of occupational illumination and illumination in the home, I wonder whether he would give information as to the illumination of test charts.

DR. ALFRED COWAN: I think that test cards should be illuminated by about 100 foot-candles, with adequately lighted surroundings. Wide variations in the intensities around this range will cause relatively small changes in the visual acuity. Also, with this amount of illumination there will be less fatigue of the patient in those instances in which considerable time is required for refraction by the subjective method.

DR. GEORGE S. CRAMPTON: Because of my experiences in connection with the Illuminating Engineering Society, I recommended this contact on the part of ophthalmologists as a pleasant and scientifically profitable one, as both groups are interested in light as a medium through which to gain their end-results. It would seem advisable to go slowly in adopting the high intensities on test charts, as recommended by some, as the fatal element of glare can readily become a menace under these intensities and defeat the purpose of a reposeful refraction.

DR. ALFRED COWAN: Of course there must not be glare, and Dr. Crampton is correct in his statement that glare will cause fatigue; but there should be no glare in the illumination of test cards if the surroundings are properly lighted. It is important that the room in which a highly illuminated test card is used should be diffusely illuminated in the proper proportion in order to eliminate glare.

EXPERIMENTAL AND CLINICAL STUDIES OF CATARACT. DR. I. S. TASSMAN.

Moerner in 1894 described the chemical analysis of the crystalline lens and its reactions. Later investigations have shown the chemical changes which take place in the crystalline lens during the development of senile cataract. Following this, the results of the investigations concerning the changes which take place in the calcium content in the lens and blood serum of patients with cataract, the cholesterol content, the permeability of the capsule of the lens and the oxidation-reduction system of the lens have been described under the following groups:

1. Experimental cataracts, such as those produced by feeding animals either naphthalene, lactose or galactose.

2. Cataracts produced as the result of either light rays or heat.

3. Cataracts of so-called endocrine origin.

4. Vitamin deficiency, particularly vitamin C deficiency, and its relationship to the development of cataract.

5. Immunologic studies, with reference to cataract.

DISCUSSION

DR. C. A. CLAPP: The first type of cataract that Dr. Tassman has mentioned, which I wish to discuss, is the glassblowers' or radiation cataract. Dr. Tassman has mentioned Professor Vogt's work along this line. There has been much controversy as to whether the opacities which develop are the result of heat or of the infra-red rays. I was privileged to see Dr. Vogt give rabbits radiation with the infra-red rays at 9 a. m., and at 2 p. m. on the same day the lenses showed a general clouding. It did not seem that heat was a factor in the production of the opacities because of the fact that since the rays were passed through a water filter only a slight feeling of warmth was experienced when one held the hand in the ray.

I have also had a series of rats on the vitamin G deficiency diet of Langston and Day, but my rats did not show cataract as frequently as theirs did. In regard to those rats which did show lenticular opacities, I felt that in every case one could detect damage in the capsular epithelium. I am inclined to believe that this damage, by altering the nutrition to the fibers of the lens, caused the opacities.

The results of the experiments on the series of rats placed on the diets of lactose and galactose were also disappointing, since all the rats died before opacities developed.

While Dr. Tassman did not mention cataract following the use of dinitrophenol, I believe this type of cataract also is caused by damage either to the capsular epithelium or to the epithelium of the ciliary processes, either one of which may disturb the nutrition of the lens. Dally has stated the belief that so-called "dinitrophenol cataract" is due not to dinitrophenol but to dinitronaphthol, which may occur as an impurity, since no cases of this cataract have been observed in France, but all have originated in the United States. Experimentation on animals seems to be without results, since so far my animals have failed to show any opacities of the lenses.

p_H AND BUFFERS IN THEIR RELATION TO OPHTHALMOLOGY. DR. J. B. FELDMAN.

This paper will be published in full in a future issue of the ARCHIVES.

ROYAL SOCIETY OF MEDICINE, LONDON, SECTION OF OPHTHALMOLOGY

Feb. 12, 1937

MR. W. H. MCMULLEN, O.B.E., F.R.C.S., *President*

THE ACTS OF CLOSING AND OPENING THE EYES. SIR ARTHUR HALL, F.R.C.P.

Oculogyric movements have been said to occur in 20 per cent of the cases of parkinsonism. In these attacks there is usually a temporary upturning of the eyes, which first became known when

epidemic encephalitis appeared. There are at least two other forms of ocular attack: one, a fixation of the gaze, and the other, inability to open the eyes. The extraordinary feature of the latter form is that when the patient wishes to open the eyes, light tactile contact with the lid breaks the spell, and the eye opens. In the third stage of the disease the patient is unable to lift his arm high enough to touch his eyelid, and then when a friend is asked to touch the lid the eye opens. In the oculogyric attack the eyes do not always turn directly up; sometimes they turn up to one side. In none of these attacks is there any marked wrinkling of the skin of the forehead.

There are certain regulations which must be observed by the eyes when a pattern is formed for binocular vision: The two eyes must act together; they must always be in a state of attention, and the pupils must be kept clear of the eyelids. Sir Charles Bell gave, in 1823, a description of what happens to the eyes when they are closed. An investigation of the eyes of 223 persons in sleep showed that the eyes were up in 65 per cent, straight forward in 29.5 per cent, down in 3 per cent and to one side in 2.5 per cent. Among children, the proportion of those in whom the eyes were up was not so great as the proportion among adults, and among a few blind children examined the proportion was still smaller. It is therefore clear that the eyes may be in almost any position during sleep.

The movements of the eye when the lids are closed may be divided into three types, according to the quantitative ascent of the eyeball: (1) very high; (2) less high, the lids being held, and (3) movements in which the eyeball does not turn up at all and may turn down. Among 900 cases, in 58 per cent the movements were of type 1; in 32 per cent, of type 2, and in 10 per cent, of type 3.

Congenital Retinal Fold. Mr. Adrian Caddy.

A school girl aged 13 showed a retinal fold in the right eye, extending from the disk outward toward the periphery of the fundus, in the direction of 8 o'clock. The eye was slightly divergent, and no blood vessels were seen. This condition of congenital retinal fold has been extremely well described by Miss Mann in the December 1936 issue of the *British Journal of Ophthalmology*, and a similar condition was described by Mr. Jameson Evans at a meeting of the Midland Ophthalmological Society last summer.

DISCUSSION

Miss Ida Mann: Unless the drawing shown is faulty, I do not think this case belongs to the group I described. In all the cases described by me there were on top of the fold branches of the retinal vessels which came up from the surface and, in some cases, ran along its sharp edge. I suggest that the condition in the present case is primarily an abnormality of the vitreous, which has become secondarily attached to the surface of the retina.

Mr. Malcolm Hepburn: I have observed a case of a condition similar to this in appearance, the scar tissue being definitely choroidal inflammatory scar tissue, and the fibrous tissue going backward in the

form of a band. In this case, however, the tissue is not so evidently scar tissue, as there is not so much proliferation of pigment as in my case.

UNUSUAL OPACITIES IN THE CORNEA. MR. E. F. KING.

A married woman aged 53 came to the hospital a few weeks ago for new reading glasses. Routine examination of the cornea revealed peculiar opacities. They were of two kinds: first, feather-like opacities, chiefly in the temporal half of each cornea, just beneath the epithelium, and, second, deeper and more circular opacities, which were beneath the level of the feathery ones. Apart from early senile cataract, the patient was in good health. These findings suggested hereditary nodular corneal degeneration.

DISCUSSION

MR. T. HARRISON BUTLER: A case almost identical with this in appearance was presented by Dr. Bamford at a meeting in Derby. In the present case all the opacities seemed superficial, and I think that the condition is chronic epithelial thickening.

Book Reviews

Trachoma. By A. F. MacCallan, C.B.E., M.D., F.R.C.S. Price, 21 shillings. Pp. 225, with 20 illustrations, the majority in color. London: Butterworth & Co., Ltd., 1936.

This book replaces in a more extensive form MacCallan's well known monograph "Trachoma and Its Complications in Egypt," published in 1913. The author's wide clinical experience as director of the Egyptian ophthalmic hospitals and his further activity in the campaign against trachoma since his return to London render him peculiarly fitted for the writing of a treatise on this disease.

Perhaps the most distinctive feature of the monograph is the great number of drawings in color which illustrate the chapters on the clinical manifestations and sequelae of trachoma. Unquestionably they add greatly to the value of the book, particularly the value to ophthalmologists in relatively trachoma-free areas in which opportunities for seeing all stages of the disease are limited. The majority of the drawings were obtained by MacCallan from Cuénod and Nataf in Tunis and from Wilson in Cairo.

The second chapter of the book is devoted chiefly to descriptions of the clinical stages of trachoma according to the classification suggested by MacCallan a number of years ago and now employed by most trachomatologists. Two new conceptions are advanced: 1. The author now describes, on the basis of the observations of Michail, a lymphocytic type of trachoma of stage I, characterized by a generalized subepithelial infiltration with lymphocytes, without the formation of follicles. 2. The term "bleb-like excrescences" is introduced to describe the expressible so-called follicles, resembling sago grains, of stage II*a*. The author states that these excrescences are not true lymphoid follicles but cysts of meibomian glands due to blockage of ducts by pressure of subepithelial infiltration and cicatricial contraction. Accordingly, the gelatinous materials expressed from the so-called follicles of trachoma of stage II*a* are to be considered not follicular contents, as is generally believed, but débris, sebaceous material, and inflammatory cells from these cysts. In the reviewer's opinion this conception needs additional evidence to substantiate it.

In the chapter on differential diagnosis the conditions which bear a resemblance to trachoma are grouped as common, uncommon, rare and tropical. It is interesting that in the first group are placed such conditions as acute conjunctivitis with follicles and chronic conjunctivitis with corneal vascularization resulting from former interstitial keratitis, while among uncommon conditions are listed spring catarrh and inclusion conjunctivitis. On the basis of the reviewer's experience in three sections of the United States, the relative frequency of these conditions in this country would seem to be reversed.

MacCallan emphasizes in this chapter, as well as repeatedly in other parts of the book, the diagnostic value of slit lamp examination of the cornea for the detection of the early signs of trachomatous pannus. He states that pannus is trachoma of the cornea and that its presence

can be detected as soon as the earliest diagnosis of trachoma of the conjunctiva can be made.

MacCallan apparently considers that no real advances have been made in the therapy of trachoma. In his chapter on treatment most of the new therapeutic measures suggested in the past few years are mentioned but are not recommended. Mechanical expression of follicles and daily swabbings with a 10 per cent solution of copper sulfate are considered most valuable. In cases in which there is secondary bacterial infection, treatment with silver nitrate is recommended. The author states that long-continued oral or tonsillar sepsis has a distinctly bad effect on the disease. The surgical treatment of the cicatricial sequelae of trachoma is described in detail. Diagrams have been omitted purposely.

Sixteen pages of the chapter on epidemiology are devoted to a discussion of the etiology. The work of recent investigators is cited in detail. It is to be regretted that in referring to several reports by joint authors MacCallan names only the senior investigators. No conclusion as to the etiology is drawn, but the virus theory seems to be regarded with the most favor.

The final chapter is devoted to the history of trachoma from the earliest Egyptian records to the present time. Considerable space is devoted to the ophthalmic campaign in Egypt from 1903 to date.

PHILLIPS THYGESON.

Transactions of the Ukrainian Leonard Hirshman Memorial State Ophthalmologic Institute. Volume 3. Edited by E. B. Rabkin. Pp. 178. Kharkov, U. S. S. R.: Ukrainian State Medical Publishing Board, 1935.

This volume consists of thirteen papers written by the pupils and co-workers of Prof. Peter P. Prokopenko on the occasion of the fiftieth anniversary of his medical, scientific and pedagogic activities. The papers are in Russian, but each one is followed by an abstract in English. The volume opens with a greeting to Professor Prokopenko from his pupils and is followed by a short biography of Professor Prokopenko, written by I. O. Merkulov.

The first paper, by E. B. Rabkin, is concerned with the polychromatic plates for the diagnosis of color blindness, which are discussed elsewhere, in a book review in the ARCHIVES.

I. I. Merkulov and S. U. Minkin report on the relation between the intracranial and the intra-ocular pressure in two patients. They found no correlation between the intracranial and the intra-ocular pressure, even when the intracranial pressure was acutely increased or decreased.

The results of an investigation on the adaptation to darkness in cases of optic neuritis and of choked disks showed that in the former the adaptation is disturbed, while in the latter group it is normal except in cases in which the condition is very advanced (I. I. Merkulov and A. G. Bereshaja).

K. I. Babenko investigated the ocular tension in the healthy eye when one eye (of a rabbit) was traumatized. If the trauma caused pain the tension in the healthy eye was always increased. The introduction of cocaine locally or procaine hydrochloride retrobulbarly

delayed the rise in tension, and the rise was less marked. General narcosis (induced by chloral hydrate) completely inhibited the rise of tension in the normal eye.

N. O. Tchepurin found that an injury to one eye lowered the adaptation to light in the uninjured eye. Removal or evisceration of the injured eye was followed by a return to normal of the adaptation to darkness in the sound eye.

D. M. Natanson, E. V. Torgovitzkaja and E. S. Gottlieb, in an article which is not suitable for detailed report here, report favorably on the use of tuberculin for tuberculous conditions of the eye.

N. I. Medvedjev reports a new operation for trichiasis. He makes an incision along the intermarginal space of the upper eyelid and then transplants a strip of bulbar conjunctiva into this space. This method is not applicable in cases in which the trichiasis is accompanied by curving of the tarsal plate.

H. B. Stepanova, N. S. Asarova and A. E. Goldfeder report on a biologic method for the isolation of Bacterium granulosis.

A. E. Goldfeder describes an interesting operation for trachomatous pannus. The conjunctiva is removed at the affected part of the cornea, but three or four conjunctival bridges are left. Then a flap of cartilage taken from the auricle is placed in the peridectomized space under the conjunctival bridges.

There are also papers on the reduction of diseases of the eye in the Ukraine, by E. B. Rabkin and I. N. Miller; on the constitution of the eye and its relation to the general body constitution, by A. I. Dashevsky; on methods of studying fatigue of the eyes, by A. I. Dashevsky and R. B. Zaretzkaja, and on elementary education of school children in the hygiene of the eye, by R. B. Zaretskaja. W. F. DUGGAN.

Physician, Pastor and Patient; Problems in Pastoral Medicine. By George W. Jacoby, M.D., Past President of the American Neurological Association and the New York Neurological Society. Price, $3.50. Pp. 390, with 20 illustrations, chiefly portraits. New York: Paul B. Hoeber, Inc., 1936.

Jacoby, long a successful practitioner and teacher of neurology, has previously written two books for nonprofessional readers—"Child Training as an Exact Science," treating of the relationship between the physician and the teacher, and "The Unsound Mind and the Law," on the relationship between the physician and the jurist.

In the present volume his purpose is to discuss without bias the relations between medicine and religion—the physician and the clergyman. He believes that the antagonism between the two has been overemphasized for centuries and that the real conflict has been between the narrow-minded and dogmatic of either profession, whereas the liberal-minded of each now agree on many points and are cooperating to bring about better health—physical, mental and moral.

The first sections of the book, on the physician's calling and on religion and the patient, comprising 180 pages, are devoted to a historical résumé of the development of medicine from its beginning and to an account of the past and present medical beliefs and practices

of Judaism, Christianity and Islam, as well as of the numerous religions of the East.

The following 150 pages on vital problems confronting the physician and the clerygman treat in detail of the following moot subjects: contraception and abortion, birth control, the questionable sanity of the suicide, divorce, the psychology of the criminal, sterilization, sex education, insanity, euthanasia, vivisection and professional secrecy.

The concluding section, of 40 pages, has as its topic "Where Medicine and Religion Join Hands in Everyday Life."

An enormous quantity of information is assembled in this volume, and debatable matters are discussed impartially, so that the reader, having before him the data and the arguments pro and contra, may form his own conclusions on the many points whereon the clergyman and the popular opinion of the past, as expressed in civil law, are wofully at variance with the physician in his present day beliefs.

WARD A. HOLDEN.

Der Augenbefund in seiner diagnostischen und differentialdiagnostischen Bedeutung bei Tabes dorsalis, Lues cerebrospinalis, multipler Sklerose. By Carl Behr. Price, 7.60 marks. Pp. 60, with 26 illustrations. Berlin: S. Karger, 1936.

In this treatise Behr, one of the most competent and successful investigators in the bordering fields of neurology and ophthalmology, deals with the ocular symptoms of some diseases in which the former frequently not only precede the other neurologic signs and symptoms but are decisive for differential diagnosis. First there is discussed the anatomicophysiologic basis of the ocular symptoms of cerebral origin, especially the anatomic structure of the optic nerve and the pathways transmitting the different stimuli to the pupil. The typical Argyll Robertson phenomenon, which occurs solely in tabes and is therefore an unequivocal sign of this disease, presents a combination of absence of reaction to light with miosis, increased convergence reaction and absence (or decrease) of the sensory and psychic dilatation reflex. Absence of the light reflex without the other aforementioned characteristics is met with not only in tabes but in other intracranial diseases. Absence of the light reflex combined with a tonic, i. e., protracted, convergence reaction, occurs in tabes as well as in epidemic encephalitis. This pupillary disorder resembles an incomplete absolute rigidity of the pupil and is found in oculomotor paresis before the fibers innervating the sphincter iridis are completely paralyzed, so that there is no pupillary reaction to light but a tonic, though sometimes ample, convergence reaction. The reviewer has seen the same behavior of the pupil rather frequently in cases of basal oculomotor paralysis, as well as in nuclear ophthalmoplegia interior, after an incomplete recovery. The pupil was dilated; its reaction to light was absent and never did return. The convergence reaction, however, which was imperfect and slow in the beginning, improved gradually until it was finally normal with regard to the amount as well as the speed of movement. Such cases are discussed at length in the reviewer's "Die Lähmungen der Augenmuskeln," in von Graefe and Saemisch's "Handbuch der gesamten Augenheilkunde," 1932. The different manner in which syphilitic and metasyphilitic diseases are produced accounts for the inexpediency or danger of antisyphilitic treat-

ment in metasyphilitic diseases. Stimulated by the good results obtained in cases of progressive paresis with malaria infection, Behr, eight years ago, started experimenting with intracutaneous injections of very small doses of tuberculin or peptone in the treatment of tabetic atrophy of the optic nerve. He got the impression that by such injections the atrophic process can be retarded, if not brought to a standstill. A rapid progression of the tabetic atrophy may be caused by antisyphilitic treatment, particularly in cases in which the atrophy is combined with papilledema. The same warning holds good if dark adaptation is considerably diminished and the visual field for colors is much contracted, while the limits of the field for white may still be fairly normal. The optic atrophy in association with tabes, as a rule, precedes the impairment of vision, in contrast to the course in disseminated sclerosis. The degeneration of the nerve fibers follows the degeneration of the neuroglial tissue, by which the blood supply is gradually reduced.

In syphilis cerebrospinalis, gummatous tumors, alterations of the blood vessels or inflammation (meningitis) may bring about ocular disorders. Retrobulbar neuritis that causes a central scotoma yields to antisyphilitic treatment. Peripheral contraction of the visual field may be combined with either homonymous or bitemporal hemianoptic defects, according to whether the optic tract or the chiasm is affected. The good results of antisyphilitic treatment are sometimes transitory, since the cerebral substance may be injured by contraction of the blood supply due to secondary shrinkage of the granulation tissues. Paresis of the third nerve is frequent, paresis of its interior branches (ophthalmoplegia interior) being one of the earliest signs of syphilis cerobrospinalis. Congenital syphilis may be the cause of dystrophia adiposogenitalis in children.

In disseminated sclerosis the medullary sheaths of the optic nerve have a predilection for injury, while the axis-cylinders frequently remain intact. As a rule, sclerotic zones in the chiasm, as well as in the optic tract, cause no symptoms, while if located in the optic nerve they bring about a central scotoma, owing to the descending degeneration. A sudden attack of complete blindness or pronounced amblyopia, without the eyeground showing any pathologic changes, is one of the characteristics of disseminated sclerosis. After a short time peripheral vision returns, while the central scotoma remains for a longer period, its size gradually diminishing. Finally, normal vision may be restored notwithstanding the fact that the atrophic discoloration of the optic disk becomes more and more evident. Cocaine and epinephrine administered by tamponade of the nose may hasten improvement in vision. Patients with acute retrobulbar neuritis must be kept in bed six weeks, since the circulation of the infectious cerebrospinal fluid is quickened by physical efforts. Transitory and recurrent paresis of either individual muscles or associated movements occur frequently in cases of disseminated sclerosis, particularly in cases of paresis of lateroversion associated with paresis of the sixth nerve of the same side. Jerky nystagmus in association with lateroversion confined to the eye with the paretic abducens nerve points to a supranuclear pontile lesion involving the sixth nucleus.

Behr's booklet presents so many important facts and considerations that a thorough study of it is highly recommendable.

A. BIELSCHOWSKY.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

Honorary President: Prof. F. de Lapersonne, 217 Fauborg St. Honoré, Paris.
President: Dr. P. Bailliart, 66, Boulevard Saint-Michel, Paris (6e).
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the President, Dr. P. Bailliart.

International Ophthalmologic Congress

Secretary: Dr. E. Marx, Costzeedijk 316, Rotterdam, Holland.
Place: Cairo. Time: Dec. 8-14, 1937.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33, Welbeck St., London, W., England.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. P. E. H. Adams, 6, Holywell, Oxford.
Secretary: Dr. Thomasina Belt, 13, Mitchell Ave., Jesmond, Newcastle-on-Tyne.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. Lohlein, Jena.
Secretary: Prof. A. Wagenmann, Heidelberg.

Midland Ophthalmological Society

President: Dr. T. E. Ashdown Carr, 34, Charnwood St., Derby, England.
Secretary: T. Harrison Butler, 81, Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.
Time: Oct. 1, 1937.

Ophthalmological Society of the United Kingdom

President: Mr. Gordon M. Holmes, 9, Wimpole St., London, W. 1.
Secretary: Mr. J. D. M. Cardell, 27, Weymouth St., London, W. 1.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Oxford Ophthalmological Congress

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Time: July 8-10, 1937.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warszawa.
Place: Lindley'a 4, Warszawa.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Ransom Pickard, 31, East Southernhay, Exeter, England.
Secretary: Dr. A. Rugg-Gunn, 35, Harley St., London, W. 1.
Time: June 11, 1937.

SOCIÉTÉ FRANÇAISE D'OPHTHALMOLOGIE

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. F. Berg, Uppsala, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. William L. Benedict, 102 Second Ave., S. W., Rochester, Minn.
Secretary: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Place: Atlantic City. Time: June 7-11, 1937.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Frank E. Burch, 408 Peter St., St. Paul.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Palmer House, Chicago. Time: Oct. 10-15, 1937.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Harry Friedenwald, 1212 Eutaw Pl., Baltimore.
Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.
Place: Hot Springs, Va. Time: June 3-5, 1937.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.

SECTIONAL

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. S. Schmidt, 107 E. Walnut St., Green Bay.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. James J. Regan, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Road, Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. A. J. Ridges, Walker Bldg., Salt Lake City, Utah.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: Salt Lake City, Utah. Time: May 24-27, 1937.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. L. H. Klemptner, 509 Olive St., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, Virginia Mason Hospital, Seattle.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. A. H. Pember, 500 W. Milwaukee St., Janesville, Wis.
Secretary-Treasurer: Dr. W. H. Elmer, 321 W. State St., Rockford, Ill.
Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each month.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Robert Griswell, 707 Washington Ave., Bay City, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. L. H. Hohf, Yankton, S. D.
Secretary-Treasurer: Dr. J. C. Decker, Francis Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
Secretary: Dr. O. M. Marchman, Medical Arts Bldg., Dallas, Texas.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. O. B. McGillicuddy, 1908 Capitol Band Tower, Lansing, Mich.
Secretary-Treasurer: Dr. Maurice C. Loree, 120 W. Hillsdale St., Lansing, Mich.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. W. Beals, Weber Bldg., DuBois.
Secretary-Treasurer: Dr. C. W. Beals, Weber Bldg., DuBois.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Edna M. Reynolds, 227 16th St., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Walter L. Hogan, 750 Main St., Hartford.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Time: May, November.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. B. H. Minchew, 701 Elizabeth St., Waycross, Ga.
Secretary-Treasurer: Dr. Edward S. Wright, 1001 Medical Arts Building, Atlanta, Ga.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. R. Dillinger, French Lick.
Secretary: Dr. Frederick V. Overman, 705 Hume-Mansure Bldg., Indianapolis.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. James A. Downing, 406 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
Place: Des Moines.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. D. R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Secretary-Treasurer: Dr. Walter E. Camp, 1918 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Edward S. Murphy, Northern Pacific Hospital, Missoula.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. C. Coulter Charlton, 124 S. Illinois Ave., Atlantic City.
Secretary: Dr. H. L. Harley, 124 S. Indiana Ave., Atlantic City.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Walter S. Atkinson, 168 Sterling St., Watertown.
Secretary: Dr. Marvin F. Jones, 121 E. 60th St., New York City.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. J. M. Lilly, 302 Old St., Fayetteville.
Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.
Place: Charlotte. Time: October.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Trygve Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 514 6th St., Valley City.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. Nathan Bolotow, 108 Waterman St., Providence.
Secretary-Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. E. Houston, 103 E. North St., Greenville.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. W. Potter, 601 Walnut St., Knoxville.
Secretary-Treasurer: Dr. W. D. Stinson, 248 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.
Place: Fort Worth. Time: Dec. 11 and 12, 1937.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Edwin W. Burton, University of Virginia, University.
Secretary-Treasurer: Dr. George G. Hankins, 202 Medical Arts Bldg., Newport News.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. F. O. Marple, First Huntington National Bank Bldg., Huntington.
Secretary: Dr. J. E. Blaydes, First National Bank, Bluefield.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Samuel T. Hubbard, 294 State St., Hackensack, N. J.
Secretary: Dr. William F. McKim, 488 Sanford Ave., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Andersen, First-Central Tower, Akron.
Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. William C. Warren Jr., 478 Peachtree St., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Jesse W. Downey Jr., 529 N. Charles St., Baltimore.
Secretary: Dr. Mary L. Small, 18 W. Read St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Thurber LeWin, 112 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. G. Henry Mundt, 30 N. Michigan Ave., Chicago.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. A. D. Ruedemann, 2020 E. 93d St., Cleveland.
Secretary: Dr. Fred W. Dixon, 1029 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. A. B. Bruner, 629 Euclid Ave., Cleveland.
Secretary: Dr. M. W. Jacoby, Hanna Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Andrew Timberman, 21 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Claude S. Perry, 40 S. Third St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. A. W. Davidson, City National Bank Bldg., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 720 Medical-Professional Bldg., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Hugh L. McLaurin, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. Maxwell Thomas, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. William Fowler, 1424 Maccabee Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. A. W. Greene, 148 Barrett St., Schenectady.
Secretary-Treasurer: Dr. Joseph L. Holohan, 317 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
Secretary-Treasurer: Dr. Charles R. Lees, 306 W. Broadway, Fort Worth.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 500 Metz Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Henry C. Haden, 1914 Travis St., Houston, Texas.
Secretary: Dr. George C. Farrish, 1625 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. J. C. Daniel, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. H. B. Davis, 1101 Grand Ave., Kansas City, Mo.
Secretary: Dr. Byron Black, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. K. C. Brandenburg, 110 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. Isaac H. Jones, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Arthur M. Zinkham, 815 Connecticut Ave., Washington.
Secretary: Dr. E. J. Cummings, 1835 I St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Hychener, 130 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Thomas A. Judge, 735 N. Water St., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 208 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Minneapolis Ophthalmological Society

Chairman: Each member in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

Montgomery County Medical Society

Chairman: Dr. A. G. Farmer, 1040 Fidelity Bldg., Dayton, O.
Secretary-Treasurer: Dr. Rome M. Webster, 663 Reibold Bldg., Dayton, O.
Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. Stuart Ramsay, 1496 Mountain St., Montreal, Canada.
Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., West, Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Secretary-Treasurer: Dr. Fowler Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. John H. Dunnington, 30 W. 59th St., New York.
Secretary: Dr. LeGrand H. Hardy, 30 E. 40th St. New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. H. Stokes, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. John S. Van Winkle, 297 Broadway, Paterson, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

PITTSBURGH SLIT LAMP SOCIETY

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Falk Clinic. Time: 4 p. m., second Friday of every month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. A. F. Bagby, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Carl T. Eber, 308 N. 6th St., St. Louis.
Secretary: Dr. W. M. James, 508 N. Grand Ave., St. Louis.
Place: Oscar Johnson Institute Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Joseph W. Crawford, 490 Post St., San Francisco.
Secretary: Dr. Russell Fletcher, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. John T. Crebbin, 624 Travis St., Shreveport, La.
Secretary-Treasurer: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. A. Veasey Jr., 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Philip B. Green, Old National Bank Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. W. J. Werfelman Jr., 725 State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St., W. Toronto.
Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. James M. Greear Jr., 1740 M St., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Ernest Sheppard, 927 17th St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.